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PEDIATRICS NUMBER

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1. The Physiological Basis of Medical Practice. 1945, p. 486.
2. New England J. Med. 235:80, July 18, 1946.

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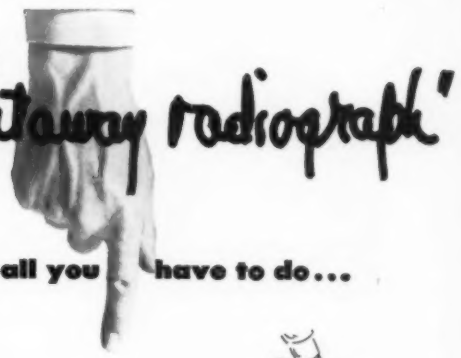

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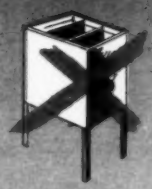

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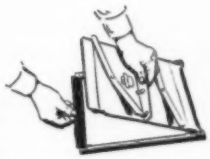

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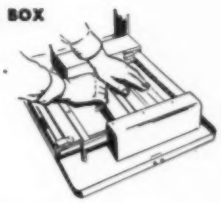

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1. Flour and Bread Enrichment, 1949-50: Prepared by The Committee on Cereals, Food and Nutrition Board, National Research Council, Washington, D. C., 1950.
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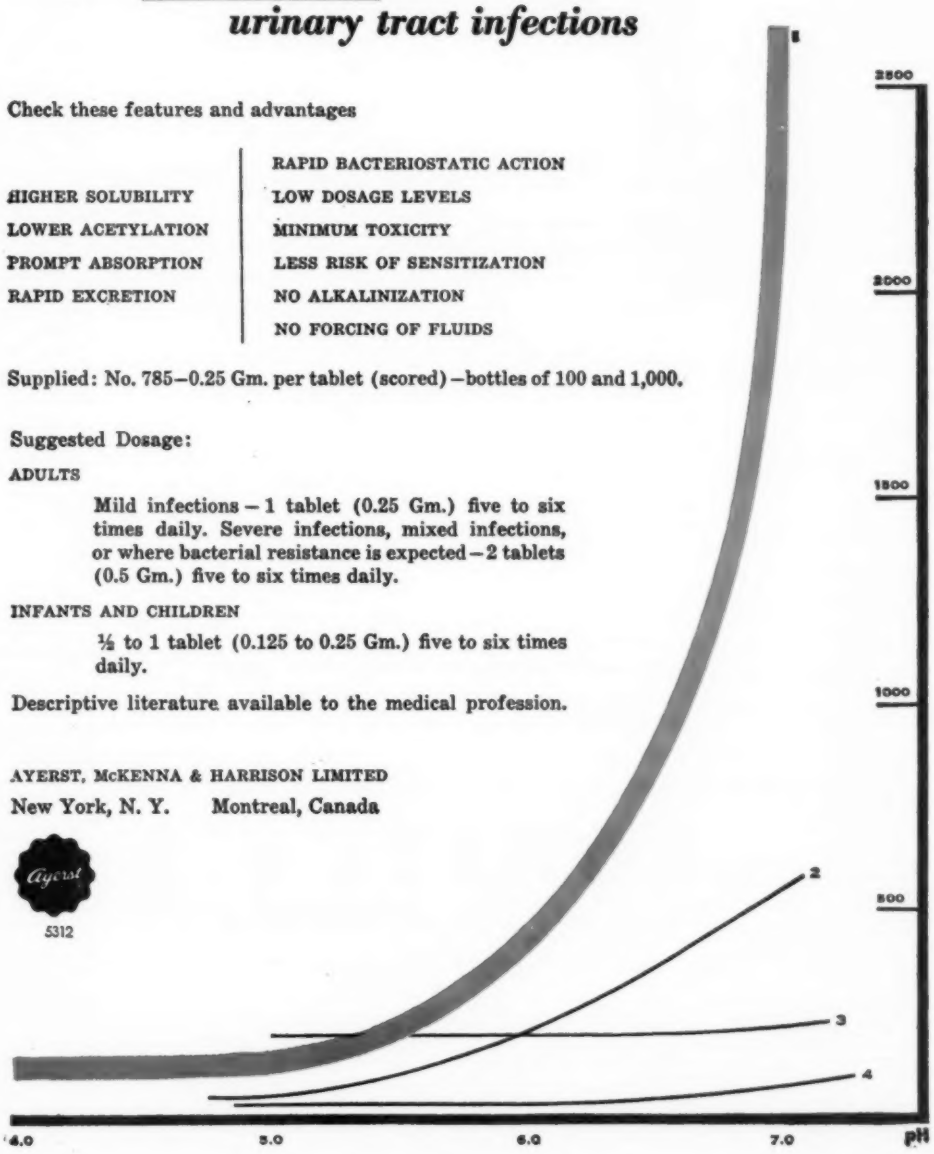

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1. Cantor, A. J., Am. J. Proctol. 3:204-210, (Sept.) 1952.

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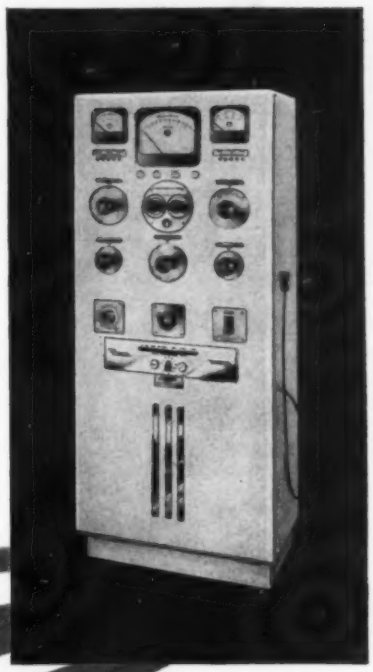

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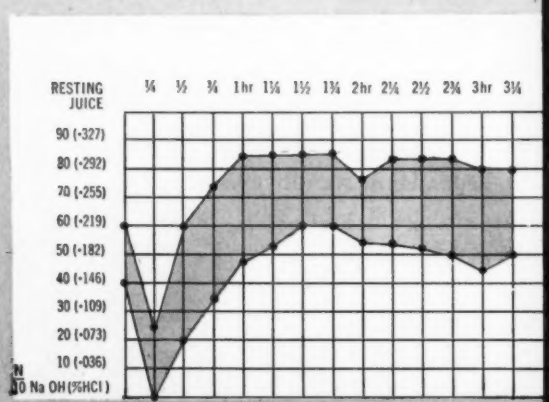

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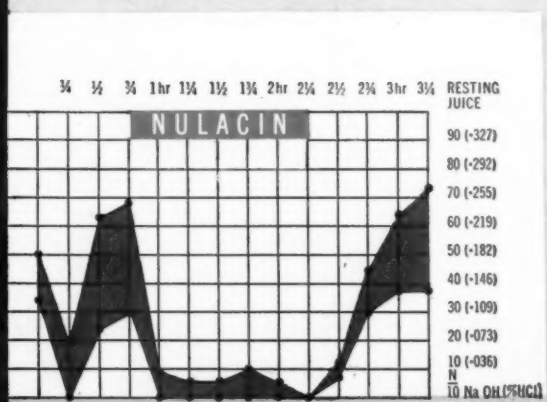

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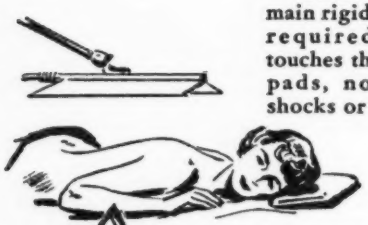

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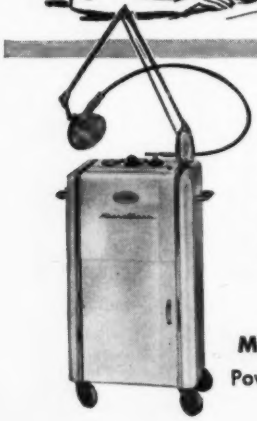

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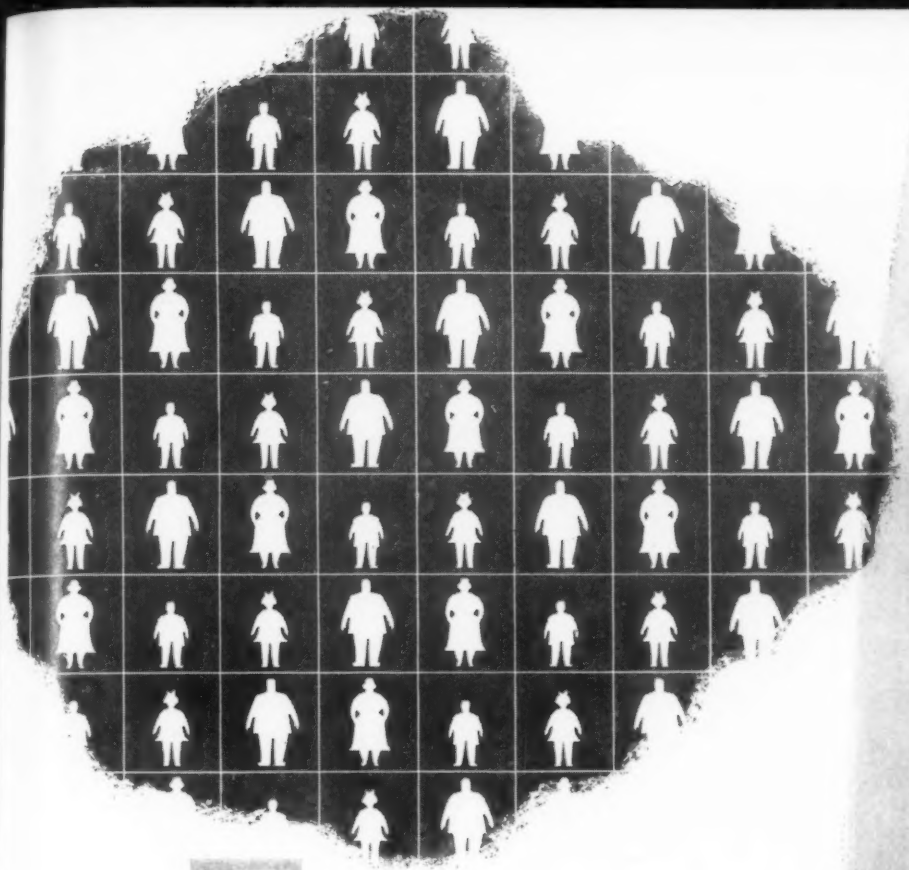

A 4-oz. glass of orange or grapefruit juice half an hour before lunch and dinner can materially reduce the demand for high-caloric foods, and enable the obese to adhere to their dietary regimens more satisfactorily. Citrus is particularly appealing because it is a "natural". non-medicinal, appetite-appeaser.

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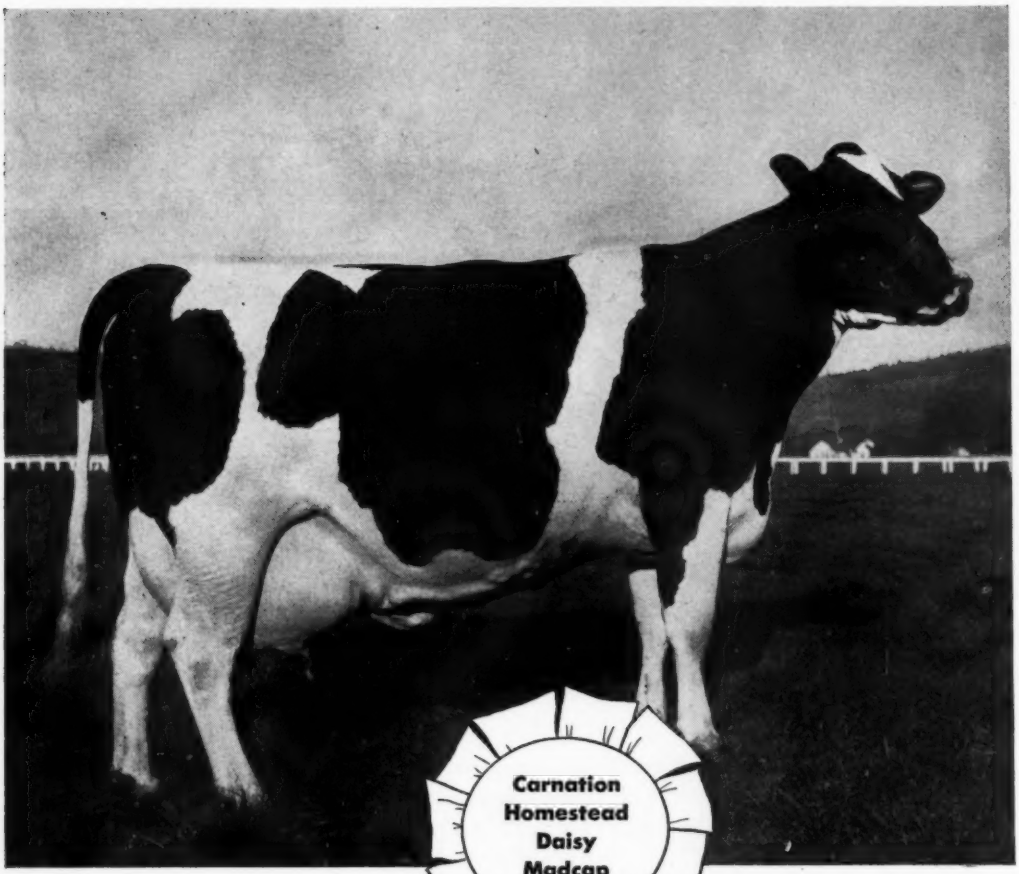

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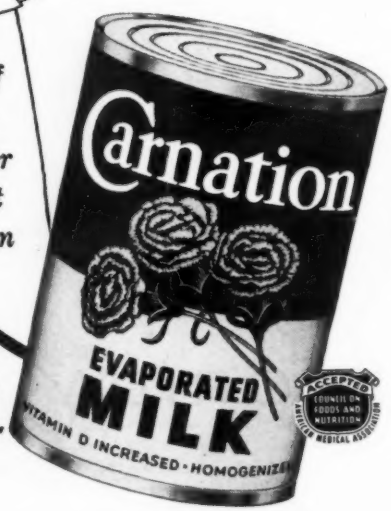

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- **its freedom from side actions**

AN ORIGINAL RIKER RESEARCH PRODUCT

obtained from

Rauwolfia serpentina

in mild and moderate hypertension . . .

Rauwiloid

What it is:

Rauwiloid represents an alkaloidal extract of the root of *Rauwolfia serpentina*, found chiefly in India. The hypotensive action of this drug has long been known, but a purified, standardized dosage form heretofore has not been available. In the form of Rauwiloid, all the advantageous features of the drug are provided in a reproducible alkaloidal mixture for the treatment of hypertension. Each batch of Rauwiloid is biologically tested in dogs for its effectiveness in producing drop in blood pressure, bradycardia, and sedation.

What it does:

The hypotensive action of *Rauwolfia serpentina* is of moderate intensity.[1] It is not apparent for several days after therapy is initiated and does not attain its maximum extent for weeks or even months.[2] When therapy is stopped, the hypotensive effect persists for some time.[3] Coincidentally with the hypotensive action, Rauwiloid produces a mild bradycardia, especially appreciated in the presence of the tachycardia which so frequently disturbs the hypertensive patient. A significant feature of Rauwiloid is the distinct sense of well-being and emotional calm it induces, as well as the prompt relief of symptoms experienced by the patient. In a series of 326 patients, drowsiness was noted in only seven.

The entire daily dose of Rauwiloid may be taken at one time, for instance upon retiring. Rauwiloid produces no undesirable side actions, even when given in excessive amounts. Dosage therefore is not critical, hence dosage calculation is not necessary. Rauwiloid is not a ganglionic or adrenergic blocking agent, and does not interfere with postural reflexes.

Available evidence shows that when a second hypotensive agent is given in conjunction with *Rauwolfia serpentina*, its effect is exerted in addition to that of *Rauwolfia serpentina*.[1] In severe hypertension, the concurrent administration of Rauwiloid and Veriloid leads to a response which may well be greater than the sum of the effects of the two drugs, suggesting true potentiation.

NOTE THESE ADVANTAGES

The desirable hypotensive action of Rauwiloid is characterized by these advantageous features. It produces

1 Pronounced subjective improvement

2 Emotional calm especially appreciated by apprehensive patients

3 Mild bradycardia, not tachycardia

4 No undesirable side actions

5 No toxic effects; there are no known contraindications

Indications:

Rauwiloid is the medication of choice in mild and moderate hypertension; in severe and malignant hypertension its synergistic properties are valuably employed in conjunction with other medication. There are no known contraindications.

Administration and Dosage:

The initial dose of Rauwiloid is 4 mg. daily, given as a single dose at night, and continued until the desired effect has been attained. Maintenance dosage at 2 mg. daily can be instituted when symptomatic and objective improvement indicates, usually after one or two months.

How Supplied:

Rauwiloid is available in 2 mg. tablets in bottles of 60, a month's supply of medication for the average patient.

in severe or resistant hypertension . . .

Rauwiloid + Veriloid®

Rauwiloid+Veriloid provides the greater hypotensive response required for controlling the symptoms and objective signs of severe or resistant hypertension. In this combination, consisting of 1 mg. of Rauwiloid and 3 mg. of Veriloid, the effect of the Veriloid is superimposed upon that of the Rauwiloid. In most patients, the response appears to be greater than simple summation of the two effects, pointing to synergistic potentiation. Furthermore, the calming influence of Rauwiloid makes it possible for more patients to tolerate Veriloid, with less likelihood of side actions, greatly expanding the applicability of the combination.

The average dose of Rauwiloid+Veriloid is one tablet three times daily, at intervals of not less than four hours, ideally after meals. This quantity may be increased to four tablets daily, especially after the response to Rauwiloid is fully apparent, which may require from 30 to 60 days.

Rauwiloid+Veriloid is supplied in bottles of 100 tablets, an average month's supply.

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6 Continuous, round-the-clock effect from once-a-day dosage

7 Hypotensive action synergistic with more potent hypotensive agents

8 No economic hardship for the patient; notably low in price for active or maintenance therapy, making it particularly advantageous in mild hypertension.

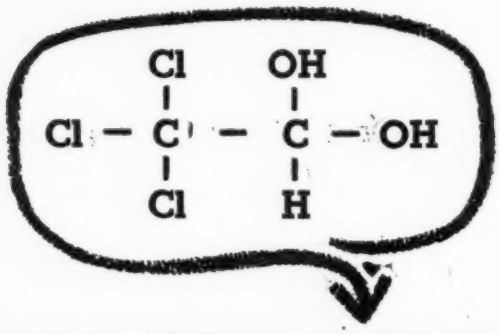
The physician will appreciate the opportunity electively to lower the blood pressure without side actions with the aim of arresting the hypertensive process.

References:

1. Wilkins, R.W., and Judson, W.E.: The Use of Rauwolfia Serpentina in Hypertensive Patients, New England J. Med. *248*:48 (Jan. 8) 1953.

2. Wilkins, R.W.; Judson, W.E., and Stanton, J.R.: Preliminary Observations on Rauwolfia Serpentina in Hypertensive Patients, Proc. New England Cardiovas. Soc., 1951-1952, p. 34.

3. Vakil, R. J.: A Clinical Trial of Rauwolfia Serpentina in Essential Hypertension, Brit. Heart J. *11*:350 (Oct.) 1949.



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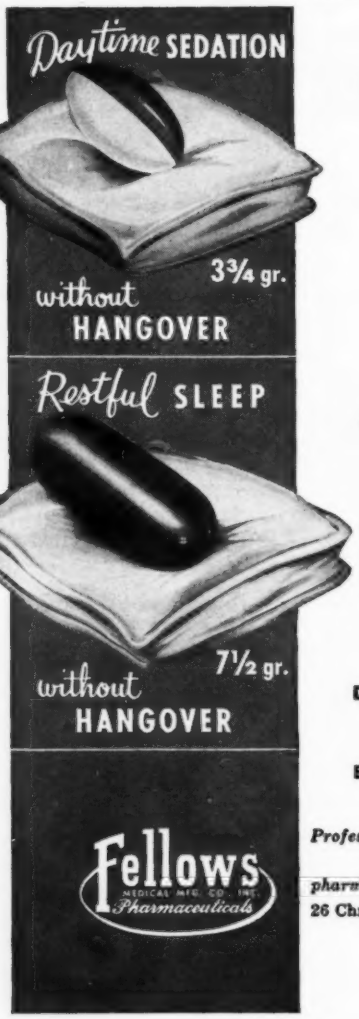

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bottles of 24's
100's

7½ gr. (0.5 Gm.) BLUE CAPSULES
bottles of 50's

3¾ gr. (0.25 Gm.) *BLUE and WHITE* CAPSULES CHLORAL HYDRATE – *Fellows*

Small doses of Chloral Hydrate (3¾ gr. Capsules Fellows) completely fill the great need for a daytime sedative. The patient becomes tranquil and relaxed yet is able to maintain normal activity.

DOSAGE: One 3¾ gr. capsule three times a day after meals.

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1. Hyman, H. T.: An Integrated Practice of Medicine (1950).
2. Rehfuss, M. R. et al: A Course in Practical Therapeutics (1948).
3. Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics (1941), 22nd printing, 1951.
4. Sollman, T.: A Manual of Pharmacology, 7th ed. (1948), and Useful Drugs, 14th ed. (1947)

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References: (1) Segal, M. S.: Springfield, Charles C. Thomas, 1950, p. 83; (2) Barach, A. L.: J.A.M.A. 147: 730-737, 1951; (3) Segal, M. S., et al.: Ann. Allergy 9: 782-793, 1951; (4) Bickerman, H. G., and Beck, G.: Personal Communication.

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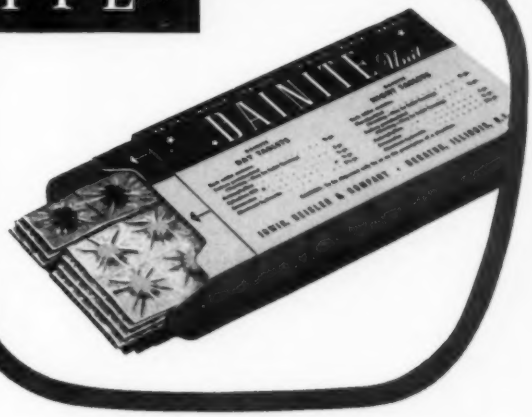
¼ gr.....	Ethyl Aminobenzoate.....	¼ gr.
2½ gr.....	Aluminum Hydroxide.....	2½ gr.
Give t.i.d.a.c.		Give at 10 P.M.



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53 Contributors

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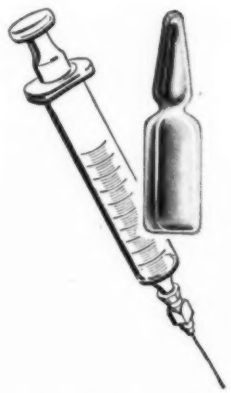
*Finnerty, F. A., Jr., and Fuchs, G. J.: Washington, D. C., to be published.



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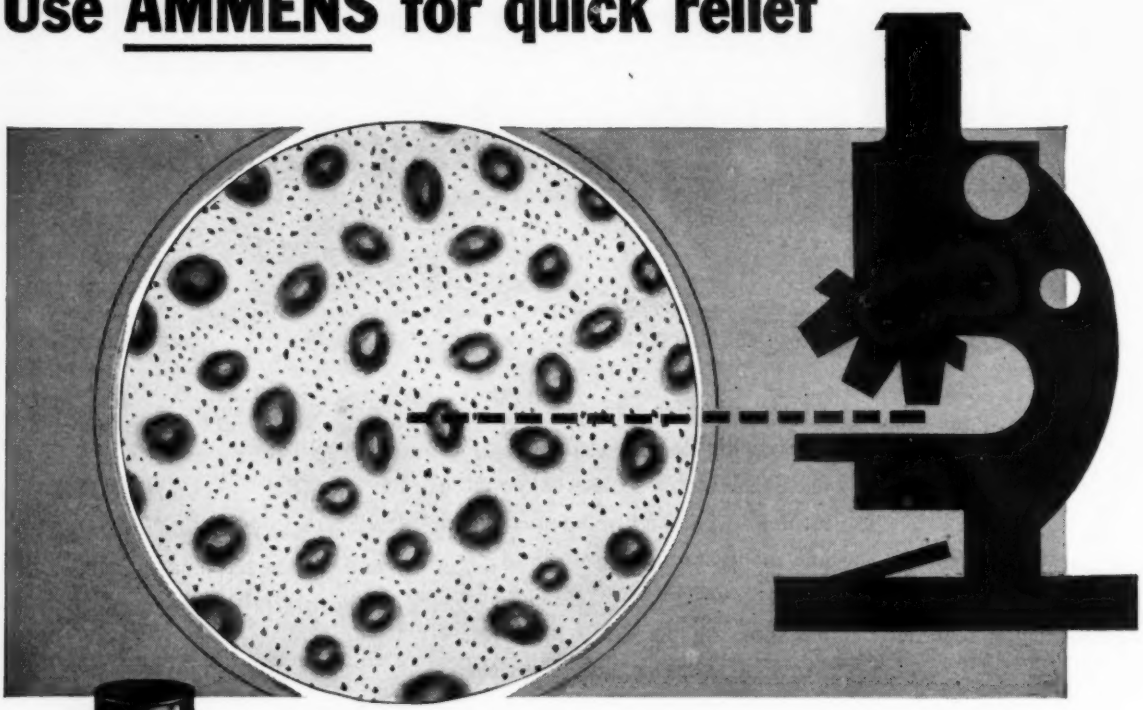

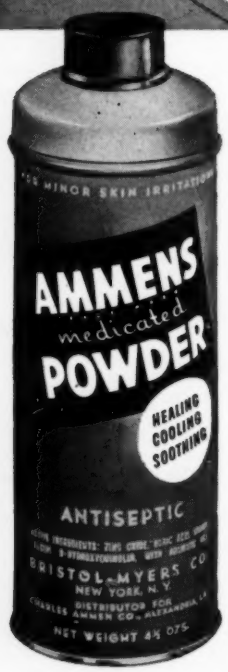

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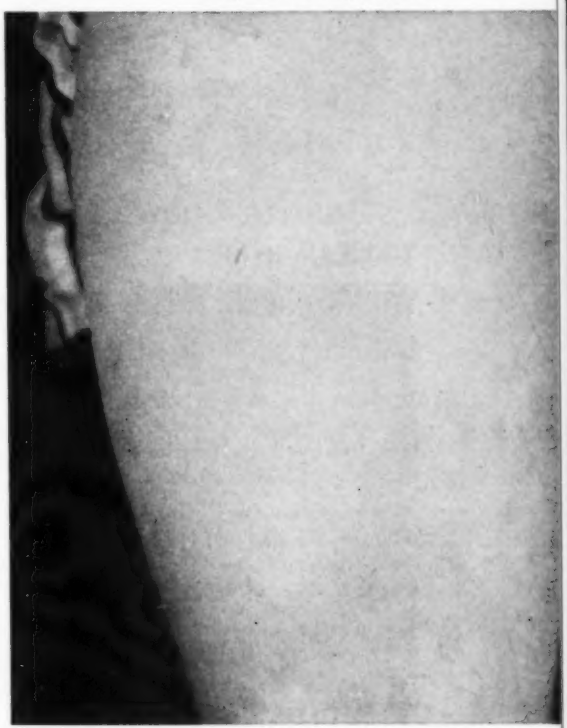

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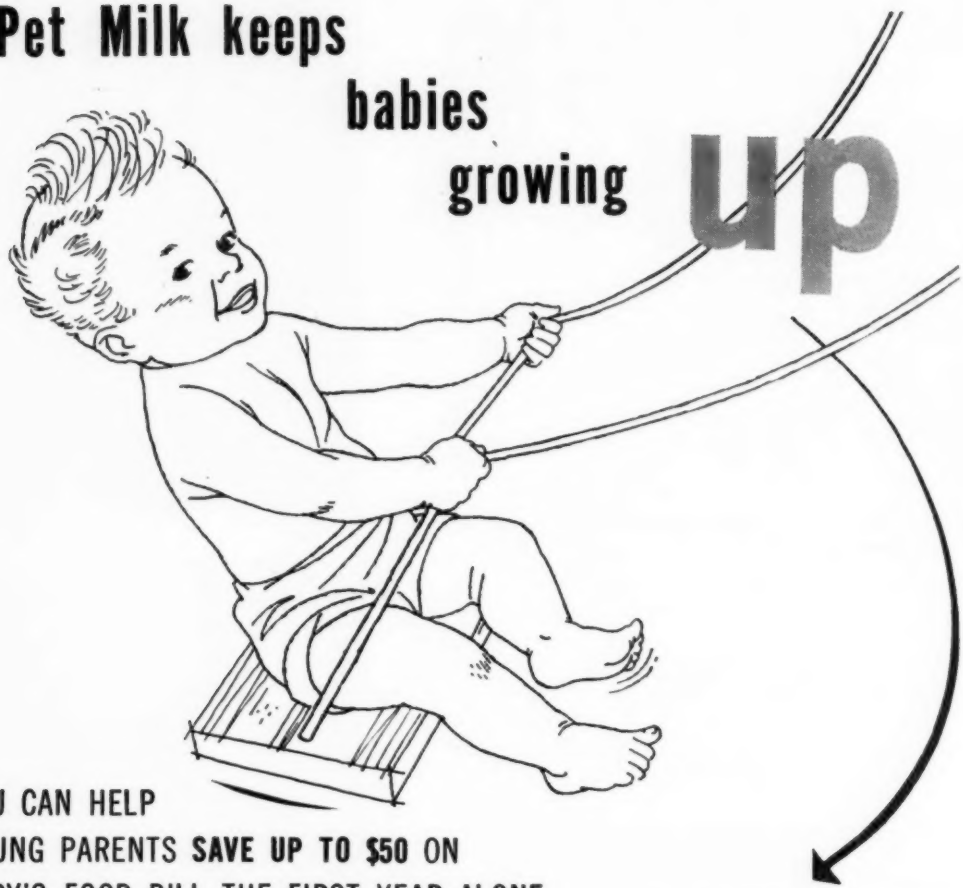

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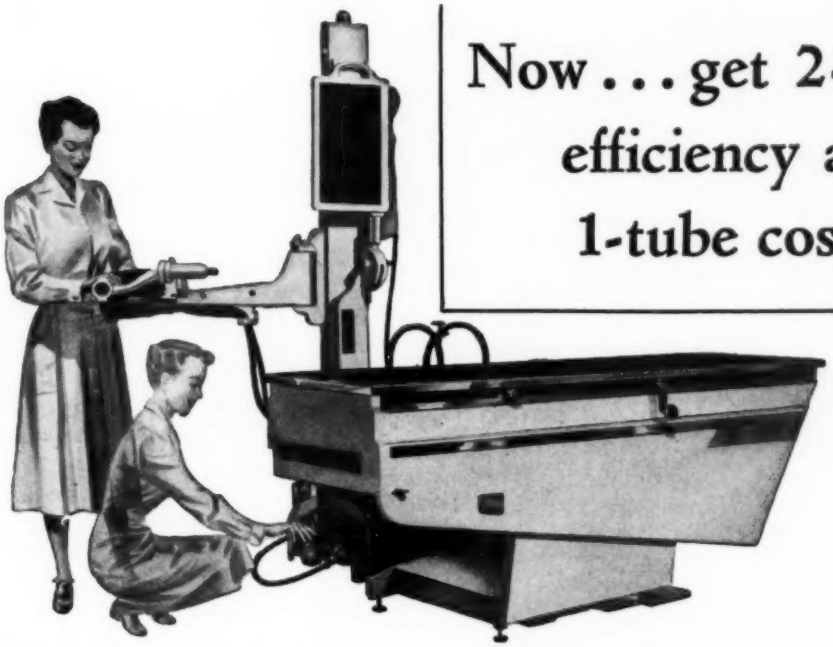

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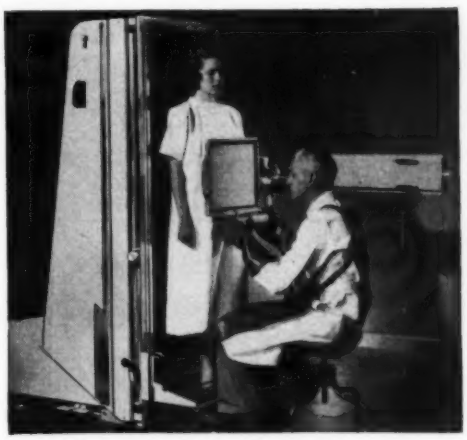

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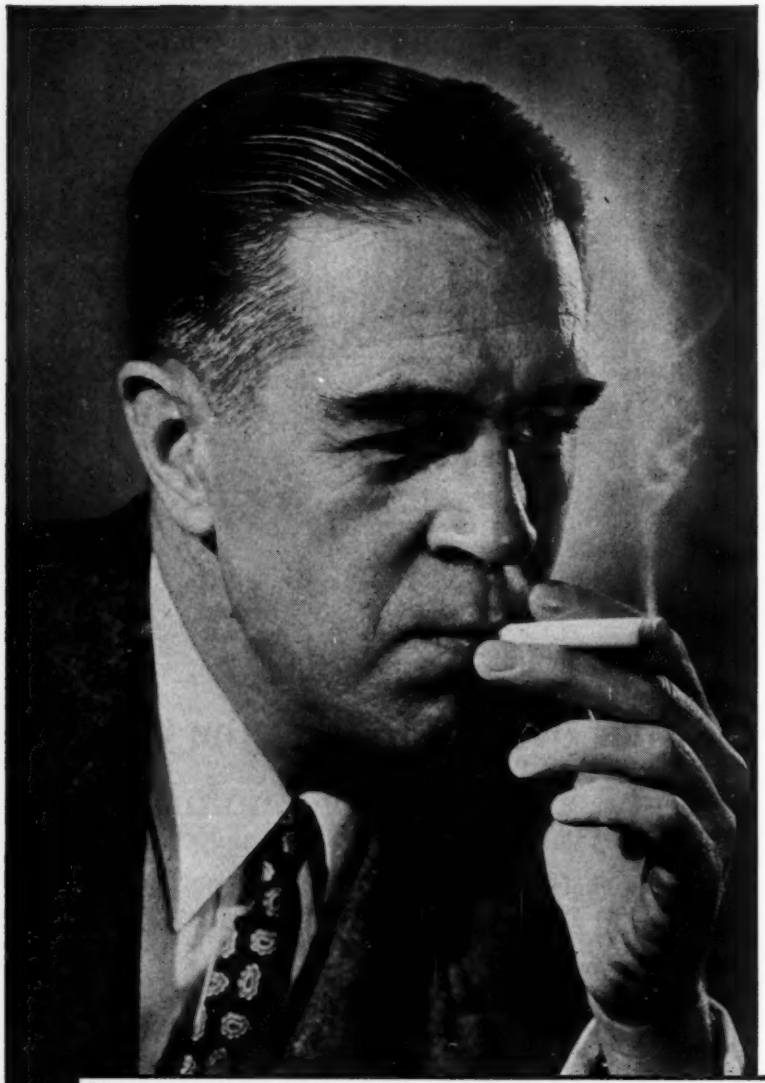

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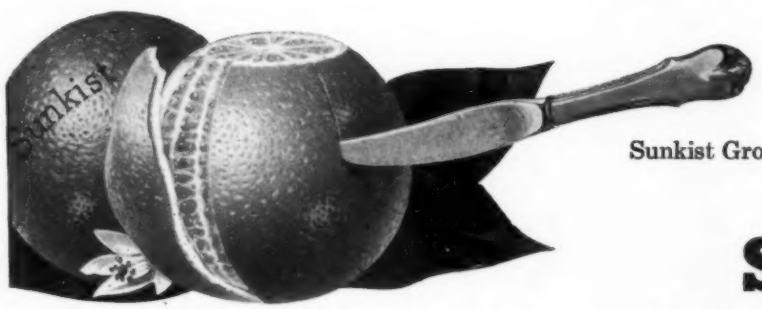

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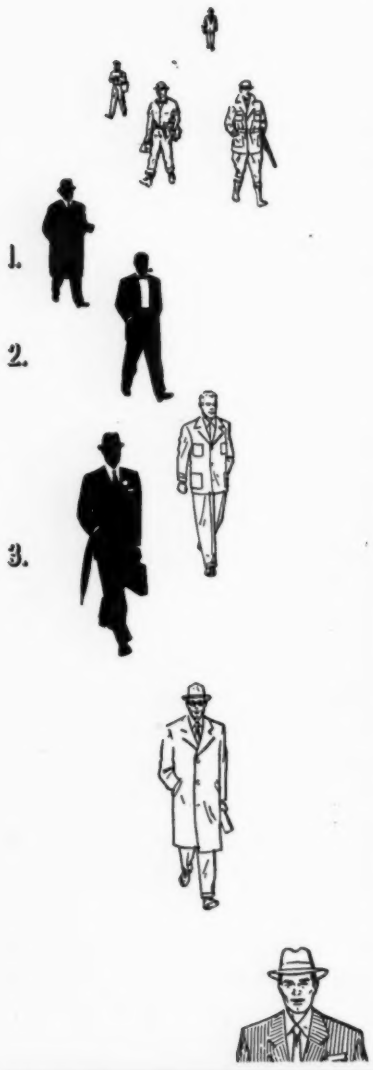

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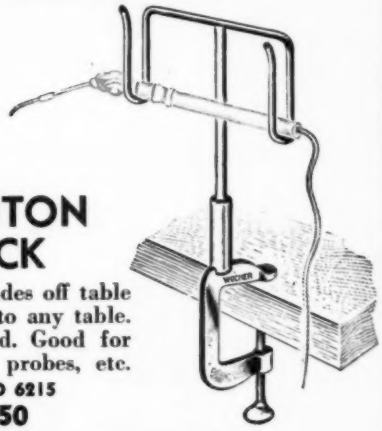

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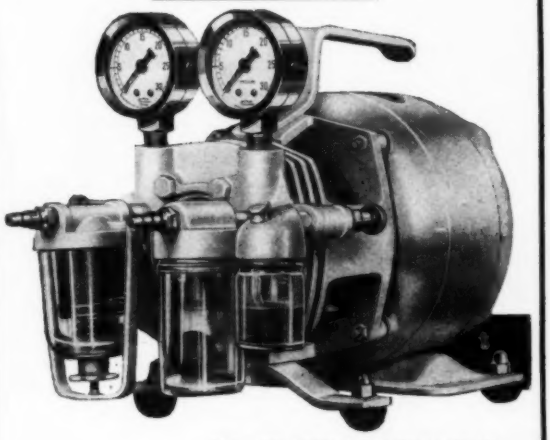

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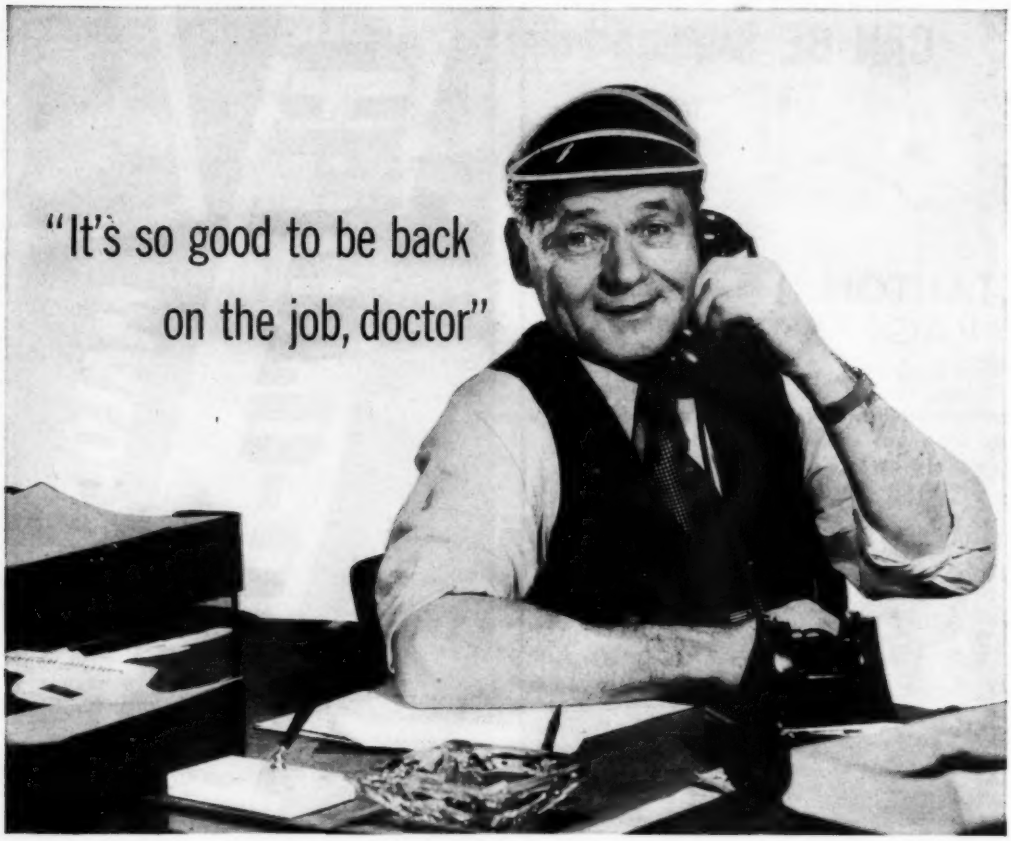

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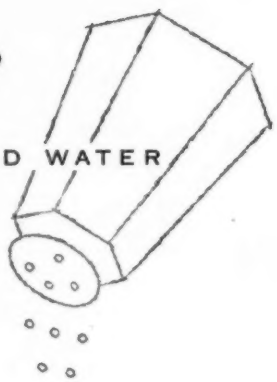

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The Use of Shock as Alterative Therapy*

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The use of shock as a therapeutic agent may seem incongruous to those who have come rightfully to regard shock as a condition to be feared and avoided. Reflection, however, will bring the realization that there are degrees of shock, ranging from the dreaded reaction which leads to death to an almost imperceptible persistent alteration in the physiology of the individual. These lesser reactions, so frequently unrecognized and seldom given consideration if noticed, may be the means of changing the health of a patient permanently; therefore, they should be an important consideration in diagnosis, treatment, and prognosis. In order to enable us as osteopathic physicians to think together on this subject it seems necessary to examine the philosophic basis for the use of shock therapy before it is discussed directly.

PHILOSOPHIC BASIS FOR SHOCK THERAPY

As philosophy and science develop more and more along dynamic and functional lines, it becomes apparent that a constant examination and re-evaluation of therapy in all of its applications is necessary if the conscientious physician is to give his patients the best possible consideration. The progressive physician of today cannot practice in the static empirical and authoritarian way most medicine, osteopathic and otherwise, has been traditionally taught and practiced. He must, in the light of modern thought, regard each patient as a unique individual whose reaction to stimuli has resulted in disease and who requires treatment specifically developed for him at the moment. The fact that a certain form of therapy has been comparatively successful in the past in the care of most patients does not mean that it is the best for this particular patient now. Even the most serviceable therapeutic procedure, including osteopathic manipulation, should be examined critically at every application to see if it is indicated and, if indicated, to see if its application meets the requirements of the present diagnosis. It cannot be taken for granted that the methods and technics taught and used for years will be beneficial to this unique patient now requiring treatment. With stereotyped attitudes, treatment becomes routine, directed toward disease rather than toward the patient.

We believe osteopathy to be a complete system of medicine with a sound biologic basis, so it is essential that we be careful not to apply any therapeutic agent empirically. Each treatment of whatever kind must conform to the truths underlying this biologic basis as they apply to the particular patient at the present time, not to patients in general, or to a disease we may name in our diagnosis.

When the physician, continuously alert, discovers new facts that may help him to be a better physician, he will test them with a yardstick built from osteopathic theory, his own and the profession's clinical experience, and research in the laboratory. Also he will check the yardstick in the light of known facts to see if it is accurate; and he will not hesitate to correct any error which appears in it or to add any new feature which may make it more complete. The alert osteopathic physician is willing and anxious to examine his therapeutic measures objectively and to accept improvements in their application or even to reject them if they are proved less valuable than he thought or if some better procedure appears. On the other hand he will not be swept away from sensible and proved procedures by therapies of little value or by unphysiologic and worse than useless modalities which refute or do not fulfill completely the requirements of the yardstick built upon the basic tenets of osteopathy.

With these principles in mind let us examine the theories of health and disease. Korr[1] observes that all complete or integrated philosophies of health and disease have one common attribute, namely that they affirm the unity of the individual in health and disease, and so reaches the conclusion that health is the expression of a nervous system able to maintain adequate physiologic function within its internal and external environment, and that disease is the result of the action of a nervous system unable to function healthfully in its environment.

If this conclusion is true, then health may be considered as the normal expression of a normal and adequate nervous (or neuroendocrine) system and disease the normal expression of an abnormal or inadequate nervous (or neuroendocrine) system. And the structure and function of the neuroendocrine system in health must differ from that found in disease and the change from health to disease or vice versa

*Presented at the meeting of the Osteopathic Society of the City of New York, September 24, 1952.

must require a change in the structure of that system. When persistent functional changes are manifest, changes must of necessity have occurred in structure which may be on biochemical and neurologic levels as well as on gross anatomic levels. This cannot be otherwise, since structure has been defined[2] as persistent function and function and structure are but different manifestations of the same configuration.

If the theory of Still, Speransky and the classical physiologists as reported by Korr[1] is true, it appears reasonable to assume that treatment directed toward improvement of health should consist primarily of an attempt to alter the structure-function of the diseased individual's neuroendocrine system in such a manner as to allow it to express health. Treatment of functional manifestations or mere disease symptoms appears to be only temporary in effect, unpredictable, and frequently futile, since it does not consistently make the change in structure which appears to be essential to successful therapy.

To make a diseased patient function healthfully it seems necessary to follow one or both of two courses depending upon the condition of the patient. Pathologic entities which provide a maintaining factor in the cycle of the disease process may be removed by surgery or other therapy, or, in cases which do not exhibit such pathologies, stimuli may be applied which are intended to change the structure and function of a perverted neuroendocrine mechanism so that it may function healthfully. Alterative therapy falls in one or both of these categories.

It is also recognized that the environmental factors which stimulate and cause the patient's system to react by way of a disease process may at times be counteracted by such measures as antitoxins, antibiotics, antidotes, et cetera, and the process thereby abated. Nevertheless, the aforementioned alteration of structure-function must even in these cases remain the fundamental approach to the patient with a disease. Antibiotics and the like may on occasion be valuable, but the ability of the patient to fight and resist the organisms inhibited by the antibiotics is more fundamental and useful and must be the first consideration of the physician.

Diagnosis preceding treatment for alteration of the neuroendocrine system is necessarily aimed at discovering and measuring the deviations in structure which maintain the disease process. The object of diagnosis is to find what in the patient and his environment is the basis of his specific aberration. Treatment, after the environmental factors are eliminated or modified to the optimum possible under the circumstances, must consist of an attempt to so adjust and change the patient structurally that he can adapt physiologically and healthfully to his environment. This certainly implies that each patient must have individualized diagnosis and treatment, for no two patients can be alike nor is a patient the same today that he was yesterday. Also it indicates that we can set no norm toward which we aim in treatment since each person exists in a different internal and external environment which is constantly varying. Nor does naming a disease indicate the specific treatment best for a given patient.

SHOCK AS A THERAPEUTIC MEASURE

In view of the preceding conclusions let us now consider shock as a therapeutic measure. In order to explain shock therapy I will first present some of my own theoretical considerations and clinical experiences.

Burns[3] has demonstrated and Cole[4] has confirmed her findings that when an osteopathic spinal lesion is produced there results an immediate reaction in the experimental animal, characterized among other things by segmental vasoconstriction and flaccidity of tissue in areas related by reflex arcs. This is the beginning of a pathologic process affecting these reflex areas specifically and altering the general function of the animal. The immediate reaction lasts from seconds to hours and precedes the local inflammatory reaction, or resolution to normal as the case may be, depending on whether or not the structural change or lesioning is persistent. Observation has shown, and any observing physician can verify it, that correction of lesions may produce similar vasoconstriction and flaccidity. Likewise a force which does not result in structural change recognized as correction of lesions may cause these changes. The description of shock as described by Selye[5] and in standard pathology texts resembles the reactions reported by Burns. I have therefore chosen to call the first stage of lesion production and the similar stage of reaction from any stimulus severe enough to produce such a reaction a shock reaction. As used in this paper, shock is considered to be the reaction of the body to any stimulus which causes a persistent change in structure-function.

It is evident that since persistent functional changes may result from the application of force, structural changes, local and systemic, must accompany the shock. These structural changes occur through the mediation of the neuroglandular system which itself undergoes a structural change in the process, thus perpetuating the reaction. Thus we have the means of altering structure-function and, therefore, of changing the health of the patient for better or for worse. Since it alters health, shock obviously may be not only potentially beneficial, but also it may initiate disease or complicate existing processes.

It was as the result of a number of clinical events in my early practice that I came to recognize shock as a therapeutic measure; the writings of Burns[6] and, later, those of Speransky[7] and Selye[5] have given rational substantiation. In the early 1930's I wrote an article on the control of nosebleed by means of shock produced by manipulation. I experienced spectacular and unexpected results not only in the treatment of hemorrhage by manipulative shock but also in the management of trophic ulcers and other obvious conditions. Unfortunately I also produced some unfavorable reactions such as hemorrhage. However, I began to analyze my successes and failures and to observe the effects of shock, produced manipulatively and otherwise, in many of my cases and came to the conclusion that a considerable number of reactions, good or bad, were due to shock as it affected the structure-function of the patient. Gradually I came to the point of considering all my cases in relation to shock therapy. Paraphrasing Shakespeare, I asked myself, "To shock or not to shock?" Now in my treatment, manipulative or other, I continue to ask this question. If I decide that shock is indicated, I then ask why and how and where and when to shock. Each time I see a patient I must ask these questions again, for each time he is a new patient presenting a unique problem.

It must be remembered that shock is relative. The ability of the patient to react is equally as important as the force applied. Force causing shock adequate to meet the needs of a frail old lady will probably not affect a young adult, and the force that produces a change in structure-function in the old lady today may not do so tomorrow. Due to these and other variables, such as the state of health and disease, the technic of shock cannot, as is true of any technic, be taught arbitrarily; it must be developed from a keen and intelligent awareness of the condition of the patient at the moment and a recognition of the osteopathic principles involved. Routinized or empirical treatment meets the need of the patient only by chance.

When shock is not indicated an alternative method must be available. Such an alternative is functional corrective technic which is based on the theory that most lesions may be corrected by an accurate reversal of the physiologic motions or sequential series of motions which the part followed in arriving at the lesioned condition found on examination. It is effective in the presence of a minimum of tension and a maximum of release and relaxation which permit correction with a minimum reflex reaction from the stimulus of manipulation. This least reaction is desirable in many cases, but not in all cases, since shock reactions may be occasionally desirable in altering the health pattern. Although shock may be produced even by this functional or indirect technic carefully applied, the resulting reaction is not nearly as frequent in occurrence nor as violent as is the shock produced by the usual technics employed in correction of lesions, namely the thrusts or heavy pressures which are intended to move structures directly into proper positional relationships.

Shock as a therapeutic agent is to be employed only when indicated, just as cesarean section or other elective surgical or medical procedures are to be employed only when certain indications exist. When indicated shock should not be avoided any more than the life-saving cesarean section should be avoided. When shock is not indicated the physiologic or functional method of releasing tensions and changing structure-function is the method of choice. Therefore, before I administer shock therapy I ask myself, "Why am I treating this patient?" In case of emergency, where life is in the balance, I seek to avoid shock therapy lest it produce the fatal or dangerous reaction which we all fear. On the other hand I may use shock in an emergency, such as cessation of breathing during surgery, in an effort to save life, risking the harm that shock may do, directly at once or indirectly later, in the development of a perverse neuroendocrine pattern which will need to be dealt with later.

If palliation or relief alone is indicated, I consistently try to avoid shock. However, now and then there may be a case, such as some congestive headaches, requiring it. In therapy for the improvement of posture (orthopedic and/or prophylactic) in a healthy individual or one approaching health during convalescence from any acute or chronic disease, it would seem unwise to apply shock, intentionally or otherwise, lest the resulting neuroendocrine pattern express its activity as disease instead of health. This unfortunate event may and, I believe, does occur for this reason, even though the total structural condition may be improved by the process.

When after careful diagnosis I decide that a perverted neuroendocrine mechanism is producing a disturbance in function known as disease and that a certain structural alteration will change the function of the neuroendocrine system favorably and that the change should be made as quickly as possible, I feel impelled to make that structural alteration with sufficient force to shock the area into a definite functional change, and to apply that force if possible in such a way as to insure a persistent structural change which will maintain the new functional pattern. This sometimes justifies a thrust with considerable force. If I have diagnosed accurately and treated skillfully, the changed neuroendocrine system of the patient will then tend toward health and away from disease. In both acute illness and chronic disease I shock as soon and as much as I think necessary to alter the course of the disease processes, and I shock in a way and in an amount that experience has shown will produce and maintain favorable change. Depending on the response, shock may or may not be repeated. This alterative therapy produced by manipulation can be highly effective.

There is a special condition calling for the use of shock in an effort to alter the reaction of the patient to a more healthful process. Where local tissue conditions are such that manipulation incident to correction seems impossible or unwise, shock may be applied to the indicated area without the usual corrective effort to produce the persistent structural improvement. Shock need not be corrective to produce a persistent alteration of neuroendocrine reaction which changes the health of the patient. It needs only to be a sufficient stimulus, in this case a thrust or prolonged pressure on a reaction area to change the structure-function of the patient's neuroendocrine system.

Shock may be psychic, emotional, chemical, or physical, and the use of any one or all of these forms may be therapeutically useful to the physician. However, it has been pointed out that the most accessible and most easily changed portion of most disease processes is that part of the somatic component known as the osteopathic spinal lesion and that treatment of this lesion is the most scientific approach to therapy known. Therefore, the physician who recognizes the importance of using the patient's inherent defense against disease applies shock as alterative therapy most often on one or more spinal segments in an effort to change them anatomically and physiologically. Usually when he needs a change in his neuroendocrine response the patient with a disease is shocked in an area of the spine related anatomically to the disease process, although shock applied in distant related parts may produce the desired change. Similar changes may also be produced by therapies other than manipulation, but manipulation has the advantage of being more specific and controllable than other modalities. If applicable in the case, shock by manipulation seems, for these reasons, to be the method of choice when an alterative is required in treatment.

It is hoped that this discussion will direct attention to the necessity of continuously re-examining the fundamental processes and thinking by which we practice, to the end that we may be aware of how routine, standardized, or careless treatment of any kind, including manipulation, may adversely affect a

patient. Still more it is hoped that this discussion will bring the realization that specifically produced shock by manipulation may be relatively harmless to the patient and clinically useful to the physician. Therefore, if properly understood and used, shock may become a valuable therapeutic agent for those who apply the osteopathic concept in their practice.

1219 Sixth Ave.

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Lower Nephron Nephrosis*

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Burbank, Calif.

INTRODUCTION

Lucké introduced the term "lower nephron nephrosis" in 1946.[1] His original contribution correlated observations on a large group of cases of varied etiology which manifested certain common clinical and pathologic features. The syndrome has recently assumed greater importance and the term popularity because of increased incidence related to numerous cases of military trauma and widespread use of sulfonamides and blood transfusions with attendant increased risk of reactions.

Lucké originally felt the pathologic changes of lower nephron nephrosis were limited to the lower nephron or distal tubule. Others, including Allen,[2] feel both proximal and distal tubules are involved in so-called lower nephron nephrosis. Allen considers Lucké's term a misnomer and prefers the older designation "hemoglobinuric nephrosis." He comments, ". . . although the literature on the subject has, in these recent years, become voluminous, we seem not to have been brought appreciably closer to a solid understanding of these dramatically oliguric or anuric kidneys than we were a decade ago."[2]

ETIOLOGIC FACTORS

The present literature includes a wide variety of unrelated etiologic factors which may cause the peculiar symptom complex to be described, and which appear to cause sufficiently characteristic histopathologic renal changes to warrant their segregation into a definite group now known as lower nephron nephrosis. Much of the study material originated from the many cases of "crush syndrome," in which trauma was recognized as the chief etiologic agent. In this group are included crushing and compression injuries, which cause liberation of myohemoglobin and other products of muscle disruption. Such cases were particularly numerous in recent war casualties, affording a stimulus for intensive study. Large numbers of cases were soon recognized which displayed the same general clinical picture and renal changes, but which appeared totally unrelated with regard to the etiologic background. These now include incompatible blood transfusions,[3] reaction to sulfonamides,[4] rapid hemolysis by the use of distilled water during operative procedures such as transurethral prostatectomy,[5] ureteroplacental hemorrhage,[6-8] heat stroke,[9] high altitude anoxia, black water fever (Plasmodium falciparum),[10] mushroom poisoning,[11] and burns.[12] The picture of lower nephron nephrosis is frequently associated with acute yellow atrophy of the liver, particularly that produced by poisoning by carbon tetrachloride,[13] chloroform, Avertin, and arsenic. (Some writers also include alkalosis and poisoning by the photograph developer, hydroquinone, and pyrogallic acid. The findings associated with these agents, however, are open to discussion and probably should not be included at this time.)[2]

According to Lucké, the common denominator of all these apparently unrelated etiologic agents is the presence of *destruction of tissue or of blood,* and this would appear to be an important feature in all irrefutable cases described.

CLINICAL PICTURE

Although some variation in the clinical picture is seen, depending on the etiology, the essential features are quite similar. Shock or a shocklike state is usually described as the first phase, and some writers postulate its presence even though it is not recognized at the time. Usually this period of shock is successfully treated and within 24 hours the patient often appears to be in good condition. The second phase, that of oliguria, soon follows, the urinary output dropping below 500 cc. daily, and within 48 hours there may be complete anuria. That urine which is passed for a day or two may be smoky or frankly bloody, at which time a positive benzidine test is obtainable. Thereafter, usually no gross abnormality is noted. Albuminuria is always demonstrable. The presence of the heme compound (commonly responsible for the positive benzidine test) usually cannot be demonstrated after the third day, but the albuminuria is persistent throughout the course of the disease. As a rule, microscopic examination of sediment shows the presence of erythrocytes, but it may not. Always one finds granular and often pigmented casts. The acidity is characteristically high. Until recently, the specific gravity was reported regularly as low as 1.010. Recent writers[13] believe that a normal or high specific gravity is more characteristic of the oliguric phase, the lower figures being seen with the onset of diuresis.

Associated with the renal shutdown is a rapid rise in the nonprotein nitrogen of the blood which sometimes exceeds 300 mg. per 100 cc. There is like-

*Presented at the teaching Sessions of the Fifty-sixth Annual Convention of the American Osteopathic Association, Atlantic City, N. J., July 17, 1952.

wise a rise in the blood potassium and a decrease of alkali reserve. Moderate early elevation of the blood pressure is characteristic. With the initial period of shock the blood pressure usually is below normal, but by the following day will have regained a normal level, and with the rising nonprotein nitrogen a blood pressure of 150/90 can be expected. Death may ensue as early as 10 days after onset, but if this does not occur, spontaneous diuresis may develop at any time within the first 2 or 3 weeks, usually followed by recovery, complete physiologic restitution occurring over a period of several months.[14]

PATHOLOGIC OBSERVATIONS

At autopsy certain gross features are regularly seen in the kidneys. A variable amount of swelling and increase of weight of these organs is observed, the total renal weight varying from slightly over the normal of 300 grams to 500 or 600 grams. The capsules strip readily with obvious bulging of the swollen cortices. The surfaces appear pale and, on section, the parenchyma is wet and edematous. The pallor of the cortices stands out in sharp contrast to the marked injection of the medulla.

The microscopic criteria vary to some degree depending upon the observer, but there are certain features which appear to be common to all descriptions. The first of these is the lack of glomerular abnormality. Although a few cases have been reported in which evidence of glomerular ischemia is cited, these appear to be in the minority.

A second common feature of the microscopic description is the presence of *heme casts* within the distal portions of the tubules. The number of casts seen is extremely variable, in some instances creating a striking picture and in others forming an unimpressive part of the histopathology. No definite dilatation of the tubules proximal to the obstruction is reported. Thus it appears evident that the degree of tubular obstruction by heme pigment is insufficient to account for the oliguria.

Some degree of *tubular epithelial damage* is regularly seen, but there is disparity in the degree and even the location. According to Lucké, there is little doubt that the greatest amount of tubular degeneration is limited to the distal segments of the nephrons including the ascending limb of Henle, the distal convoluted tubules, and the collecting tubules. However, this limitation is not held by all authorities; for instance, Allen[2] and McManus[15] emphasize the frequency of cases in which damage to the proximal tubular epithelium is nearly as prominent a feature as that seen in the distal segment.

Associated with the tubular damage and the precipitation of heme pigment there is uniform agreement in regard to a *stromal inflammatory reaction*, apparently as a result of tubular epithelial degeneration followed by herniation of the irritating content into the peritubular stroma or into a nearby thin-walled vein. If a vein is thus eroded, thrombophlebitis usually results, further exciting the stromal reaction.

In addition to the heme casts, one often sees protein casts in the proximal nephrons which are not specific to this disease but nevertheless form a prominent part of the picture.

PATHOLOGIC PHYSIOLOGY

Reflecting the state of chaos of the etiology, the terminology, and even the microscopic features of this disputed disease, the pathologic physiology is as yet only theoretical. These theories, however, have considerable value in determing the therapy.

Since the presence of the heme compounds (hemoglobin or myohemoglobin) is stressed and actually forms the basis of the descriptive entity, its importance, from the standpoint of functional renal disturbance, should be considerable. However, we have no direct evidence as to this importance. We know that hemoglobin, having a molecular weight of approximately 70,000,[1] comparable to that of serum albumin, is not normally present in the glomerular filtrate. On the basis of animal experimentation and observation, it is felt that in lower nephron nephrosis glomerular permeability is increased, resulting in a widening of the pores permitting the passage of this substance of high molecular weight. The reason for the altered glomerular permeability is debatable, but may be due to renal vasoconstriction as a result of the presence of large amounts of hemoglobin within the circulating blood. Vascular constriction, due to shock, may also play a part. Myohemoglobin, resulting from a breakdown of muscle, has a molecular weight of 17,500 and readily passes through unaltered glomerular capillaries. It is felt, however, that those conditions leading to the liberation of myohemoglobin from injured muscle also may cause renal vasoconstriction and an increased permeability of the glomerular filters.[1] This altered permeability is demonstrated clinically by the almost invariable occurrence of proteinuria.

The cause for the precipitation of the heme compounds in the tubular lumina is subject to great dispute. Many have felt that precipitation occurs only in an acid medium. Some experiments by Oliver[16] would suggest that the necessary isoelectric point for coagulation is not encountered until the filtrate reaches the distal tubules, at which point cast formation may take place. There is yet considerable speculation as to whether the heme compounds are toxic, causing the tubular epithelial degeneration, or the precipitation is the result of tubular disintegration.

Much experimental work and theorizing have been done with regard to the possibility of nephrotoxic substances arising from injured muscle or the breakdown of tissue. Adenosine triphosphate has been mentioned as one of the nephrotoxic substances which may be liberated. An increased concentration of the adenosine compounds has been demonstrated in blood returning from ischemic muscle. It has been suggested that if adenosine compounds are liberated *slowly* into the blood stream, the liver is able to detoxify them and thus spare the kidney subsequent damage.[1] If such hypothesis is correct, it may help to explain the great variation in the occurrence of lower nephron nephrosis following trauma, depending upon the degree and duration of muscle ischemia and the functional capacity of the liver.

"One of the key physiologic problems in need of clarification is the mechanism of oliguria and anuria."[2] Many authorities, including Lucké, feel that glomerular filtration is adequate, but that all or most of the filtrate is reabsorbed across necrotic tubular epithelium into peritubular capillaries. This is also offered as an explanation for the reported frequent low specific gravity, indicating failure of concentrating ability by the tubular epithelium. However, this explanation breaks down when one recalls the fact that the *proximal* tubule is responsible for the concentration of

glomerular filtrate and that these authors postulate that the proximal nephron is structurally intact. Furthermore, recent observers suggest that the oliguric urine is more frequently of normal or high specific gravity. Allen feels that, despite a lack of supporting evidence, glomerular ischemia constitutes the most reasonable explanation for the anuria. He ties this in with the clinical observation that shock results in a decrease of renal blood volume to one-tenth, one-twentieth, or even less of the normal volume. Such an argument, however, appears invalid when one recalls that the oliguria begins after the shock appears to have been treated adequately. Perhaps shock initiates renal ischemia, which is then perpetuated by the tremendous interstitial edema regularly observed in these kidneys. (The cause for the interstitial edema is not yet satisfactorily explained, and furthermore, decapsulation does not regularly hasten the onset of diuresis.[2])

TREATMENT

Strauss[14] has outlined an approach to treatment in these cases which in essence has proved to be invaluable. His conclusions were developed from the analysis of "an otherwise healthy adult man weighing 70 kg. who suddenly lost all function of both kidneys." According to his calculations, the greatest threat to life during the ensuing period of renal shutdown is the rise in serum potassium, which may lead to fatal cardiac changes before nitrogen retention or acidosis becomes threatening. Analysis of previous modalities, such as spinal anesthesia, splanchnic block, diathermy to the kidney region, x-ray radiation of the kidneys, injections of acetylbetamethylcholine, irrigation of the renal pelvis, stripping the capsule or capsulotomy of the kidney, blood or plasma transfusion, or both, intravenous sodium sulfate and hypertonic fluids, including glucose and saline solutions, massive intravenous saline or lactose solutions, peritoneal lavage, and the use of an artificial kidney, showed that although all of these methods, singly or in combination, have been tried, none appears to have had any uniform success. The following is an outline of suggested therapy:

1. During the *initial shock phase*, shock or dehydration is treated by whole blood, plasma, or saline solution.

2. *Anuric or oliguric phase.* As soon as the volume of urine output is markedly diminished, the second phase of treatment begins; 750 cc. of 15 per cent glucose in distilled water are given by vein daily, and nothing is given by mouth. Oral administration of food seems to increase vomiting. If significant emesis occurs, the amount vomited is measured and the equivalent of physiologic saline solution is added to the daily infusion of dextrose in water.

Strauss[14] stresses the fact that "During the second week of treatment great fortitude is required because of the insistent demands that the patient be drowned with large volumes of intravenous fluid. This must be resisted. If acidosis becomes clinically manifest (as well as being evident on laboratory examination), a liter of one-sixth molar sodium lactate is given, the extracellular fluid volume being thus expanded as the price for combating acidosis. . . . Electrocardiograms are made at least every other day for the observance of changes indicative of an excess of potassium." (Continuous gastric lavage may be used to decrease the potassium level.)

3. *Stage of diuresis.*—Water equivalent to the total urine volume is administered, plus 1000 cc. for insensible loss and any other losses, in addition to an amount of sodium chloride to replace that excreted in the urine on the preceding day. *Carefully matched* blood may be given if severe anemia develops. In this stage, the glomerular filtrate remains low in specific gravity, and therefore has very little ability to conserve salts. There then may develop depletion of potassium, calcium, and sodium chloride, which must be replaced. The urine volume may be in excess of 2 to 3 liters daily, thus the danger of drowning the patient is remote.

CAUSE OF DEATH

It must be emphasized that the evidence is against the assumption that these patients die as a result of nitrogen retention or acidosis.[14] A large number of cases of lower nephron nephrosis, which so frequently exhibit generalized and pulmonary edema at autopsy, have undoubtedly been drowned by excessive quantities of fluid. In those cases where pulmonary edema is not evident, potassium intoxication is thought to play a prominent part. "Available data indicate that man, unlike small animals, may survive without renal function for four or more weeks if excessive quantities of fluid are not administered parenterally."[14] Thus if the patient can be kept alive during the period of anuria until spontaneous diuresis occurs, complete functional and histologic recovery may be expected.

SUMMARY

A discussion of lower nephron nephrosis (hemoglobinuric nephrosis) is given, indicating the multiplicity of etiologic factors, which apparently have a common denominator of tissue destruction. The lack of uniformity of histologic criteria for the diagnosis is indicated, and the observation that tubular epithelial degeneration may involve the proximal as well as the distal nephrons, despite the popular term "lower nephron nephrosis." The lack of a satisfactory explanation for the chief clinical feature, oliguria, is pointed out, and the suggestion made that it may result from renal ischemia initiated by vasoconstriction during shock which is perpetuated subsequently by resulting interstitial renal edema. A method of treatment is given which avoids the usual error of drowning the patient by efforts to overcome the oliguria.

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The Salt Depletion Syndrome*

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As early as 1933, Binger and Keith[1] noted the effects of diuretics on various types of edema and in doing so, observed and commented on what today is termed the salt depletion syndrome or hyponatremia. This subject has to some degree been neglected, although it has appeared increasingly in the literature during the past several years.

A concept of health and disease which is pertinent, not only to the salt depletion syndrome, but to other disease states, postulates that a state of health represents the establishment and maintenance of the fluid matrix (extracellular fluid) within homeostatic limits. Such a situation is obtained through the various regulatory mechanisms of the body. The primary vehicle of this fluid matrix is water, and its functional capacity depends upon its electrolytic and colloidal contents, as well as upon the various gaseous contents distributed to it through the circulating blood. When this environment of the body cells is maintained within certain homeostatic limits, a state of health is present. As the environment departs from these critical limits, disease is present as a result of the fact that with altered environment the protoplasmic activity of the cell is modified, and as this activity is modified, its functional capacity is modified, which in turn produces a change in organ system function.

According to the concept presented by Gamble,[2] body fluids are divided into three compartments, the blood vascular compartment, the extracellular or interstitial fluid compartment, and the intracellular compartment. The vascular compartment represents approximately 5 per cent of body weight, the extracellular fluid compartment about 15 per cent of body weight, and the intracellular fluid compartment about 50 per cent of body weight. The units of function, namely the cells, and the intracellular compartment, representing 50 per cent of the body weight, depend upon the two lesser compartments, the interstitial and blood compartments, for maintenance of their protoplasmic integrity. With this concept as a basis, we can perhaps better understand the widespread sequence of events that follows an alteration in the electrolytic balance of the extracellular compartment in the salt depletion syndrome.

It is believed that one of the most common etiologic factors producing the salt depletion syndrome is use of mercurial diuretics in managing edema of congestive heart failure. These diuretics function predominantly at the tubular level, serving as a protoplasmic poison which interferes with the enzyme systems and therefore, with the tubular function.[3] They tend to remove disproportionate amounts of sodium, chloride, and water, causing reduction in blood chlorides, lowering of serum base, and an increase in the urinary output of sodium, potassium, calcium, and chloride. Under such circumstances, diuresis will follow. Mercurial diuretics are indicated only if the edema present is that of salt retention, not edema of water retention. Unfortunately many times the edema of water retention, which probably would respond best to the administration of digitalis with no mercurial, is not differentiated from the edema of salt retention. In the latter, there has been a shift of the sodium into the extracellular space with subsequent retention of water to allow the usual attempt to maintain a homeostatic level of the extracellular environmental fluid.

Another factor which today is contributing to the increased incidence of the salt depletion syndrome is the use of the ion exchange resins. These substances liberate sodium and postassium in acid media and imbibe or remove sodium and potassium in alkaline media. Therefore, the resins liberate sodium during a comparatively short residence in the stomach, but imbibe sodium and potassium during the longer residence in the small bowel. Because of this it is possible to feed patients a diet containing sodium and still not effect any appreciable absorption and retention of this ion. Thus use of the ion exchange resins in conjunction with average low sodium diet often results in salt depletion or hyponatremia.

Schroeder,[4] in 1949, described the salt depletion syndrome as follows: a successive depletion of urinary volume with a depression of urinary chlorides, with resultant gain in body weight, accompanied by a relative and absolute increase of sodium in the extracellular fluid. This is followed by a rise in levels of the urea nitrogen and nonprotein nitrogen contents of the blood and a fall in the level of the blood chloride and sodium. This precipitates increased cardiac rate with the appearance of peripheral edema to a marked degree. The patient then presents the following symptoms: weakness, mental confusion, apathy, edema, hypotension, dyspnea, anorexia, nausea, vomiting, muscular cramps, and peripheral vascular collapse.

It is interesting to note that the above symptoms are almost identical with those of congestive heart failure for which the patient received the therapy. When

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this symptom complex presents itself, differential diagnosis is necessary. If the patient is in the state of true congestive failure with salt retention, mobilization and excretion of sodium are indicated to correct the condition. This can be accomplished by restriction of sodium, acidification to mobilize, and mercurials to expedite excretion. However, if diuretics are utilized in the face of sodium depletion the condition is aggravated and the uremic state is worsened. MacGuire[5] notes that the risk of uremia is greater in the old age group and in those patients with minimal or potential renal disease. These people frequently alternate between a state of hyponatremia with uremia and congestive failure when under treatment. In evaluating and projecting the use of mercurial diuretics in the management of edema all these factors should be given careful consideration. Ray and Burch[3] have suggested that the mercurials be withheld if the blood urea nitrogen content is over 60 mg. per cent.

Another interesting syndrome that has long been recognized, and which symptomatically parallels the salt depletion syndrome, is adrenal cortical insufficiency or Addison's disease. These people show sodium depletion, rising blood urea nitrogen and nonprotein nitrogen, marked weakness, asthenia, hypotension, tachycardia, nausea, vomiting, hypoglycemia, et cetera. It is believed that functional disturbances of the adrenal cortex play an important role in the salt depletion syndrome; thus a better understanding of this relationship may lead eventually to more reasonable and more successful management of this syndrome.

Elkinton and his coworkers[6] have shown that with salt depletion, the contraction of plasma volume is greater than with water depletion, although the extracellular contraction is approximately the same. This coincides with the manifestations often seen in shock, resulting in decreases in plasma volume, venous return, cardiac output, blood pressure, and circulation rates. As a result there is an intracellular dehydration and an extracellular overhydration, producing cellular injury and protein loss. In this condition the importance of maintaining a constancy of the fluid matrix as an environmental medium for normal cellular metabolism is evident. When such a condition evolves, particularly with a decrease in the functional capacity of renal cells, it may be responsible for the rising blood urea nitrogen content. Further, with changes in the functional capacity of the myocardium, a decrease in renal hemodynamics will also contribute to the above change.

In the management of the salt depletion syndrome the most obvious factor is the utilization of sodium chloride for replacement of the depleted sodium ion as well as the depleted chloride ion. For a more adequate distribution of sodium a hypertonic solution should be utilized. This assures sodium replacement without excessive introduction of water and therefore allows a more adequate distribution of both intracellular and extracellular electrolytic factors without the addition of water to dilute and retain these factors in the extracellular space. At the same time mercurial diuretics should be reduced or completely withdrawn.

Elkinton, Squires, and Bluemle[7] conclude from balanced studies on edematous cardiac patients, most of whom were hyponatremic and refractory to mercurial diuretics, that in the presence of systemic sodium depletion treatment with hypertonic sodium solution may at times be successful. On the other hand, the number of failures suggests that this syndrome may be more commonly associated with factors leading to retention of water rather than to loss of salt. One of the most important factors may be an alteration in the intracellular osmolarity. For this reason some patients have responded to the combined administration of sodium and potassium, without any appreciable rise in the potassium levels of the blood stream, indicating a shift of the potassium ion into the intracellular compartment.

Squires, Crossley and Elkinton[8] further concluded that the intracellular phase shares in the body fluid abnormalities of congestive heart failure and that these intracellular abnormalities consist of potassium depletion, increased osmolarity of solutes present and overhydration. In attempt to explain theoretically some of these changes, Elkinton and Squires[9] have suggested that, in addition to changes in glomerular function and myocardial function, changes in cellular hydration may cause changes in the osmoreceptors of the posterior pituitary. These in turn would affect the antidiuretic hormone and also possibly the sensation of thirst. After prolonged administration of a mercurial, alterations in such mechanisms along with alterations in adrenal-cortical function may set a new level of total electrolytes in the body.

CONCLUSION

1. Much is yet to be learned regarding the humoral, enzymatic, and nervous factors regulating electrolyte and water balance of the fluid compartments of the body.

2. The so-called salt depletion syndrome may represent either a depletion of electrolytes or retention of water.

3. The adrenal cortex and the posterior pituitary, when altered in their functional capacity by the changes in osmolarity produced in this sequence of events, may set the total electrolytic balance at new levels and may be partially responsible for the changes observed.

4. Mercurial diuretics must be used cautiously, especially in the aged and those with evidence of renal impairment, to avoid onset of a salt depletion syndrome.

5. Management of the salt depletion syndrome at present, usually administration of sodium, is not completely successful in the majority of cases.

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Pulmonary Emphysema*

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DEFINITION

"Emphysema is a condition of the lungs where the air sacs and alveoli are abnormally distended either with or without permanent changes in their walls."[1] The state may be acute or chronic, localized or diffuse, primary or secondary to other pathologic conditions of the lungs, heart, or chest.

CLASSIFICATION

A number of classifications have been offered but the most comprehensive would appear to be this one presented by Rubin:[2]

1. Obstructive emphysema
   A. Chronic hypertrophic emphysema
   B. Bullous emphysema
2. Nonobstructive emphysema
3. Interstitial emphysema
4. Compensatory emphysema
5. Combined forms of emphysema.

OBSTRUCTIVE EMPHYSEMA

Chronic Hypertrophic Emphysema.—This is not only the most common type of emphysema but that which produces the greatest interference with respiratory function. Any condition producing distention of the lungs, particularly in the expiratory phase of respiration, can produce emphysema. Barr[3] states that "Experimentally, great distention of the lungs can be produced without damage if the pressure is quickly released, the elasticity of the normal lung causing it to return to its usual position." Even moderate distention, however, if maintained, causes permanent changes with loss of elasticity and circulatory changes. "In addition, anything which increases intrapulmonary pressure, such as coughing or straining, will advance the process."[3] Thus it may be readily seen that chronic bronchitis and long standing bronchial asthma will be the chief offenders. The impression that the respiratory effort of glass blowing and the playing of wind instruments causes emphysema has been eliminated as false. Many feel that congenital predisposition in the form of a decrease in the pulmonary elastic tissue is of great importance.

In normal health the intrapleural pressure is always negative but in emphysema it more often approaches that of the atmosphere. Loss of alveolar elasticity results in an inability to retract on expiration and thus there is an increased amount of air which keeps the lung margins against the chest wall and diaphragm. Normally the functional residual air is between 35 and 40 per cent of the total capacity but in emphysema it may reach 70 to 80 per cent.

Expiration is normally a passive, effortless process but with the distended, inelastic lungs pressing against the diaphragm and chest wall the accessory muscles of respiration must be brought into play to expel the air.

The saturation of oxygen in the blood of an emphysematous individual may be as low as 60 per cent, which is the syncopal level for the normal individual, but the patient may be comfortable because of various compensatory mechanisms.

With increased pleural pressure, negative pressure of the thorax, which aids the emptying of the venae cavae into the right auricle, is diminished and thus an increase in pressure of the lesser circulation occurs which eventually results in right ventricular hypertrophy and cor pulmonale. The large alveolus with its diminished capillary supply is of course very inefficient as far as gaseous exchange is concerned. Much of the inspired air is too distant from the remaining capillaries to add oxygen and pick up carbon dioxide. The resultant carbon dioxide increase and diminished oxygen saturation of the blood are compensated in the following manner: (1) by polycythemia, (2) by stimulation of the respiratory center (dyspnea), (3) by increased bicarbonate reserve. Owing to the chronicity of the condition the patient himself is frequently not aware of the dyspnea until it becomes advanced.

The thorax becomes greatly enlarged because of kyphosis of the upper dorsal vertebrae and elevation of the sternum. The ribs become more horizontal and the costovertebral and costosternal articulations tend to ankylose. This immobilization of the thorax still further interferes with pulmonary ventilation.

Pathology: The large, feathery, pallid lungs are usually covered with blebs and frequently cover the heart when the chest wall is opened. In hypertrophic emphysema they generally do not collapse when exposed. Diminished pulmonary capillaries, dilatation of the air sacs and smaller bronchi, and arteriosclerosis of bronchial aterioles are generally noted. Microscopically the air vesicles are torn, distended, and lack muscular fibers and capillary supply. As was previously noted, right ventricular hypertrophy is expected.

Signs and Symptoms: Dyspnea of insidious onset is the outstanding feature of this disease. Rather profound changes in respiration may occur with comparatively little distress. Because of the compensatory polycythemia less dyspnea is present at rest than would be expected. The dyspnea is most marked on exertion and is increased in rate rather than in volume.

Cyanosis can be marked and progresses with the dyspnea. "In no condition, except congenital heart disease, is there such marked cyanosis in ambulatory patients."[3] This depends upon the degree of unsaturated hemoglobin. Superimposed right heart failure may add orthopnea, dependent edema, and hepatic enlargement.

The appearance of the chest is characteristic, the marked increase in the anteroposterior diameter creating the "barrel shape." An upper dorsal kyphosis is present, the shoulders appear elevated and the neck shortened, the intercostal spaces widened, the sternum and intercostal cartilages prominent, the costal angles obtuse and, in extreme cases, almost straight. The chest is elevated as one piece and no measurable expansion is present.

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Increased resonance on percussion, occasionally almost tympany, may be noted and tactile fremitus and vocal resonance are diminished. Heart and breath sounds are feeble and rales of associated bronchial disease are generally present. Expiration may be so prolonged as to be four times that of inspiration and all areas of dullness (cardiac, splenic, and hepatic) may be obscured. The fluoroscope reveals little or no discernible diaphragmatic motion during respiration.

Diagnosis: Clendening[4] states, "I do not remember ever having made the diagnosis of emphysema as a single entity in my life: it is always associated with heart failure or something else." Clendening also tells how Cabot, the great diagnostician, related that he was quite disillusioned by statistics comparing clinically diagnosed emphysema with pathologists' findings. In 12 cases diagnosed during life only 3 were substantiated, and of 153 cases proved to have emphysema on postmortem examination only 7 were recognized during life.

Most cases of moderate emphysema are overlooked. The condition should be considered when the patient presents a history of chronic pulmonary disease and the development of progressive external dyspnea. As it progresses, cyanosis of the skin and mucous membranes and the other signs and symptoms previously mentioned appear.

Hyperillumination of the lungs and accentuation of the hilar markings as demonstrated by x-ray are helpful. Blebs and bullae often cause annular transparencies and the bronchogram with Lipiodol is sometimes diagnostic. Restriction of diaphragmatic motion and even a paradoxic movement (greater descent after normal inspiration than after maximal inspiration) is sometimes noted in advanced cases.

Diminution of the vital capacity is employed both diagnostically and prognostically. Any who have perused older textbooks of medicine will recall the method of testing which has the emphysematous individual unable to blow out a lighted candle. Circulation time and venous pressure determinations aid in distinguishing the presence or degree of associated congestive heart failure.

All authorities on the subject of emphysema are agreed that diagnosis of this condition is difficult and that many pitfalls are present which result in erroneous opinions. In the past 2 years I have seen three full-blown cases and two moderately advanced cases which had previously been labeled as having had various other pulmonary and cardiac diseases. In spite of my awareness of this condition, I have undoubtedly missed a number of early cases.

Bullous Emphysema.—Both bullae and blebs frequently appear in the previously discussed hypertrophic emphysema, but when the bullae constitute the major feature of the disease, the condition is known as bullous emphysema. A bulla comes from an area of localized vesicular emphysema within the substance of the lung but does not violate the pleura. A bleb is an interstitial emphysema within the pleura which produces a bladder-like prominence on the lung surface.

Bullous emphysema is often classified with pulmonary cysts. It appears more frequently in men than women, usually in those who are older. It may occur following the bronchial obstruction of bronchopneumonia, influenza, and other acute infections; it may be due to acute bronchiolitis from the inhalation of noxious gases; or it may appear in conjunction with fibroid tuberculosis, pneumonoconiosis, asthma, bronchiogenic carcinoma, and chronic pneumonitis.

Other terms which have been employed are: alveolar ectasia, acute vesicular emphysema, alveolar emphysema, pneumocyst, balloon cyst, and pneumatocele (expansile bullae). Bronchial or bronchiolar obstructions frequently create valvelike mechanisms which allow air to distend a group of vesicles but not be eliminated, and the bulla is thus formed. Rupture, particularly of the latter, as well as localized bullous emphysema, may cause recurrent pneumothorax. Several writers[2,4,5] refer to the unusual picture of the "vanishing lung," which is a progressive bullous emphysema.

Diagnosis is primarily radiographic when coupled with the history, dyspnea, cyanosis, signs and symptoms of spontaneous pneumothorax, and most especially the consideration of this disease as a possibility.

Recently a description of a relatively new syndrome, which should be mentioned in connection with the subject of emphysema, appeared in the literature.[6]

"Progressive bronchospastic disease resulting in emphysema of the obstructive type" is the descriptive name suggested by the author. Although various theories regarding etiology have been advanced, many similar to those of bronchial asthma, none has been established.

Although the mechanism is constriction of the smaller bronchi and bronchioles, the clinical picture differs from that of bronchial asthma. At the onset, symptoms are present only for 30 to 60 minutes after arising and consist of dyspnea and severe, paroxysmal cough productive only of small amounts of tenacious mucus. Hot beverages, vomiting, and other means of raising the small amount of sputum provide relief. With progression of the disease, increased impairment of ventilation becomes evident in the form of exertional dyspnea which is relieved by lying down. Physical signs and x-ray findings are those of early obstructive emphysema from any cause. Cyanosis, first on exertion and then at rest, follows later. Death may result from congestive heart failure or, in people from 40 to 50 years, from pulmonary insufficiency (asphyxia).

Treatment is the same as for emphysema from any cause and depends upon the stage of the disease; bronchodilators and expectorants, of course, play an important role.

NONOBSTRUCTIVE EMPHYSEMA

Senile emphysema is the most common type in this group in which the shape of the chest is the cause rather than the result of the pathologic features. Because of kyphosis, stiffness of the spine, and subsequent changes in the sternum and ribs, which all develop in the aged, the anteroposterior diameter of the chest increases. The lungs, following the course of the thoracic cage, thus are distended. The alveolar septa then atrophy. Bullae and blebs are not generally present and diaphragmatic motion is usually unhampered. Conversely to that in the obstructive type, abdominal aid to breathing is increased in this type of emphysema and, although vital capacity is diminished, it is not disabling.

INTERSTITIAL EMPHYSEMA

According to Rubin,[2] this condition is "characterized by the escape of air from ruptured alveoli into the connective tissue stroma of the lungs. The excess air finds its way along perivascular sheaths to the mediastinum, retroperitoneum and subcutaneous tissues, often escaping into the pleural and peritoneal cavities." Spontaneous pneumothorax or pneuroperitoneum may thus result.

Clinically the picture depends upon the size of the leakage area and rate of air escape. Coronary thrombosis, pericarditis, pulmonary infarction, and pneumonia might be simulated. In severe cases there is marked cyanosis, dyspnea, and retrosternal pain which radiates to the neck and shoulders, causing the patient to prefer lying flat with the chest inflexible since motion is extremely painful. Widespread subcutaneous emphysema is generally present and obscures the hyper-resonant percussion note and obliteration of cardiac dullness which would otherwise be present.

COMPENSATORY EMPHYSEMA

Loss of functioning lung volume by surgery, collapse, or congenital defects places an added load on the remaining lung tissue which then, as with other organs and tissues, attempts compensation, in this instance by the enlargement of the remaining alveoli. In addition the relative dead space created by removal of all or part of a lung tends to create a nonobstructive type of emphysema as previously discussed. Unfortunately, it often happens that thoracic surgery eventually is detrimental to the patient because of markedly diminished pulmonary function.

COMBINED FORMS OF EMPHYSEMA

Any two of the previously discussed types of emphysema may and frequently do occur in combination. One component is generally predominant.

TREATMENT

General.—General use of antibiotics is indicated, particularly the newer penicillin preparations which concentrate in pulmonary tissue, to control early bronchial infections. Aerosol antibiotics are helpful too, particularly when combined with bronchodilators. Frequent osteopathic manipulative therapy, high vitamin A and C, avoidance of exposure to inclement weather, and other supportive measures are indicated. I recently found in one case that a window-type air-conditioning unit with its dust filter and humidity-reducing quality helped a great deal.

Cough.—The usual sedatives and expectorants are employed. If the cough is primarily nonproductive, Hycodan bitartrate gives symptomatic relief. Postural drainage is recommended where bronchiectasis or copious sputum is present.

Bronchospasm.—When bronchospasm is present the usual bronchodilators employed in the treatment of bronchial asthma are indicated. Nebulized Adrenalin, Isuprel, Norisodrine, ephedrine and/or xanthines orally, aminophylline, rectally and on occasion intravenously, are all used in routine fashion. Cardalin orally holds some promise.

Abdominal Belts.—In the small number of cases I have treated all have been materially benefited by a properly fitted emphysema belt to increase intra-abdominal pressure and help in the expiratory phase of respiration. Pneumoperitoneum has been recommended for the purpose, but I have had no experience with this procedure for this condition.

Oxygen.—Because of the oxygen debt which develops, oxygen inhalations several times daily, particularly at bedtime, often give symptomatic relief for hours at a time.

Interstitial Emphysema.—Control of pain with opiates if necessary and supportive measures are generally sufficient. If pain is severe, surgical consultation with needling of the mediastinum should be employed. Tension pneumothorax is treated as from any cause.

Congestive Heart Failure (Cor Pulmonale).—Mercurial diuretics, digitalis preparations, and other usual means of treating heart failure will be necessary eventually in most cases.

Psychotherapy.—Most of these patients are chronically ill and constantly fear impending death. A proper physician-patient relationship, sympathetic understanding, and reassurance can relieve many symptoms.

SUMMARY AND CONCLUSION

The various types of pulmonary emphysema have been discussed and their mechanisms presented. Treatment has been outlined. It is felt that awareness of these conditions as clinical entities will result in more frequent early diagnosis.

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The Differential Diagnosis of Macrocytic Anemia*

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A diagnosis of macrocytic anemia is indicated when large red blood cells are found in peripheral blood smears. Bone marrow studies will substantiate the peripheral smears by demonstrating the presence of megaloblasts. The macrocytosis may be accompanied by an increase in the amount of hemoglobin, providing there is no associated iron deficiency. Once the diagnosis of megaloblastic anemia has been confirmed it is necessary to determine the etiologic factor or factors involved.

The erythrocyte goes through various stages in its development in the bone marrow and the megaloblast is one of the early stages in the maturation process. When megaloblasts occur in the bone marrow in abnormal numbers either macrocytic or megaloblastic anemia may be present. There are several theories as to whether the megaloblast found in macrocytic anemia is a normal or abnormal stage in the maturation of the erythrocyte.[1] Correct diagnosis requires an understanding of the reasons why erythrocytic maturation is altered at the megaloblastic stage of development.

An erythrocyte maturation factor or antianemic principle found in the liver is necessary for the development of the erythrocyte past the megaloblastic stage. Castle's[2] original theory stated that the erythrocyte maturation factor was a result of the interaction of an extrinsic factor found in food and an intrinsic factor secreted by the stomach and the antianemic principle thus formed was absorbed from the gastrointestinal tract and stored in the liver. At present this theory is being discarded because vitamin B_{12} is now thought to be the same as the extrinsic factor and the antianemic principle. However, some other substances such as folic acid, Will's factor, and animal protein factor apparently are necessary for the utilization of vitamin B_{12}. These substances are all found in various foods and must be ingested by the individual in certain proportions or macrocytic anemia will develop. The intrinsic factor found in the stomach is an enzyme necessary for assimilation of the antianemic principle and its storage in the liver. Thus it can be seen that any alteration in these processes will result in the production of a macrocytic type of anemia.

The megaloblastic anemia of infancy, macrocytic anemia of pregnancy, tropical macrocytic anemia, refractory megaloblastic anemia, and nutritional macrocytic anemia all may be attributed to the lack of proper dietary intake. Pernicious anemia and the macrocytic anemia following gastrectomy are due to lack of the intrinsic factor. The macrocytic anemia found in sprue, idiopathic steatorrhea, small bowel resections, fish tapeworm anemia, chronic pancreatic disease, regional ileitis, chronic dysentery, and celiac disease is caused by poor absorption of the erythrocyte maturation factor from the gastrointestinal tract. Liver disease may produce a macrocytic type of anemia as a result of faulty storage of the antianemic principle. Achrestic anemia is macrocytic in type, and is thought to be due to faulty utilization of the erythrocyte maturing factor.[4] Hypothyroidism may produce the same type of anemia, but the mechanism of production is not understood.

Obviously, with all these various possibilities, differential diagnosis of macrocytic anemia may be somewhat confusing. As a result, a complete and thorough history and physical examination of the patient are absolute necessities. It may be necessary to perform many and varied laboratory tests and x-ray examinations before the final diagnosis is made. Until the etiologic factor is isolated, therapy may be of little value.

The megaloblastic anemia of infancy occurs before the age of 1 year. The child usually appears acutely ill because of the presence of a secondary infection. Nutrition is poor, and often there is cardiac enlargement, accompanied by a functional murmur. The skin and mucous membranes may show the presence of petechiae. The blood smear and bone marrow will reveal normochromic macrocytosis and poikilocytosis; however, the mean corpuscular volume occasionally may be normal.[5] Careful evaluation of the dietary intake will in most cases substantiate the presence of a nutritional deficiency.

The adult patient with a nutritional macrocytic anemia has a history of animal protein deficiency of several years' duration. Symptoms include glossitis, peripheral neuritis, diarrhea, fatigue, dermatitis, pallor, shortness of breath, and epigastric distress. There may be some soreness of the feet and cramping of the calf muscles. This type of anemia is found commonly in the male between the ages of 40 and 80. Examination of blood smears and bone marrow will reveal abnormalities similar to those found in pernicious anemia. However, nutritional macrocytic anemia may be differentiated from pernicious anemia by the fact that with the former, hydrochloric acid is present in the gastric contents, and the icteric index and serum iron concentrations are within normal limits.[6]

Tropical macrocytic anemia has been classified as a nutritional megaloblastic anemia and is found primarily among the natives of tropical countries. This disease process appears to occur commonly in young or middle-aged women and is very frequently associated with pregnancy. Complaints are usually of glossitis, weakness, diarrhea, shortness of breath, and edema. Fever, splenomegaly, purpura, and uterine hemorrhage may occur. Blood smears and bone marrow studies are identical with those of pernicious anemia. Achlorhydria is not a common finding. There may or may not be evidence of increased blood destruction such as increased icterus index and urobilinuria.[7,8]

The term "refractory megaloblastic anemia" has been applied to macrocytic anemias that do not respond satisfactorily to intensive liver therapy. Symptoms are

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weakness and shortness of breath upon exertion. Chronic glossitis may be present. Physical examination usually reveals no significant findings. The peripheral blood and bone marrow smears are typical of any macrocytic anemia and there may or may not be an achlorhydria. The diagnosis is usually made only when the anemia fails to respond to the routine liver therapy but does improve with folic acid therapy.[9,10]

Macrocytic anemia which develops during pregnancy is thought to be of nutritional origin.[11] It occurs more frequently in multiparae under 35 years of age. Onset the anemia may be insidious or sudden and usually occurs in the third trimester. Usual symptoms of anemia may be present and also rather severe gastrointestinal symptoms such as nausea, vomiting, and diarrhea. Frequently these symptoms may be attributed to toxemia of pregnancy. The liver and spleen may be somewhat enlarged. Purpura or mucous membrane hemorrhages may be present. The blood picture may be variable, and typical of an iron deficiency anemia. The presence of megaloblasts in the peripheral blood smears will suggest the diagnosis of macrocytic anemia. However, this type of cell is not always found, and the only reliable method of diagnosis is the bone marrow study. If an increased number of megaloblasts is seen on the bone marrow slide, the diagnosis is macrocytic anemia of pregnancy. Because of extreme variability of the peripheral blood picture, a bone marrow study should always be done in a severe anemia of pregnancy.

Diagnosis of pernicious anemia may be made with ease or with difficulty. The cardinal symptoms of paresthesias, sore tongue, and weakness in the presence of achlorhydria and a macrocytic anemia make diagnosis relatively simple. However, in many cases symptomatology is extremely varied and confusing and may simulate many organic diseases. Thus, it is necessary to obtain a careful detailed history and physical examination. Beside the classic symptoms previously mentioned, there may be weight loss, lack of appetite, nausea, vomiting, diarrhea, abdominal cramps, tachycardia, palpitations, and dyspnea. The patient's general appearance may be deceiving in that few external changes are present. On the other hand, these individuals often present pallor, glossitis, and icteric sclera. Examination of the heart may reveal mitral and aortic systolic murmurs and the electrocardiograms show a depression of the ST segments with flattened or inverted T waves. These cardiac findings may be so impressive as to suggest the possibility of cardiac disease.[3] Neurologic complaints such as paresthesia and difficulty in placing the feet will be found in as many as 95 per cent of the cases. Mental signs such as irritability, depression, and memory lapses may occur in as many as two thirds of the cases.[12] Neurologic examination reveals a loss of vibratory sense and sense of coordination.

Laboratory examinations show changes consistent with pernicious anemia. Blood smears in pernicious anemia contain a hyperchromic macrocytic type of red blood cell with anisocytosis and poikilocytosis. Other blood elements such as leukocytes and platelets are reduced in number with a relative lymphocytosis. The icterus index is increased and the urine contains an increased amount of urobilin and urobilinogen. The bone marrow contains large numbers of megaloblasts. There is no secretion of free hydrochloric acid by the stomach even after histamine stimulation. This change in acid secretion is so consistent that a diagnosis of pernicious anemia should rarely be made without this finding.[3]

Surgical procedures involving the stomach and bowel, such as total or partial gastrectomy and gastroenterostomy, may interfere with the mechanism of red blood cell formation. Not every surgical case will develop a macrocytic type of anemia[13,14] but megaloblastic anemia does occur frequently and must be kept in mind as a possibility. The symptomatology is the same as that of the other macrocytic anemias, with the peripheral blood smears, and bone marrow studies showing changes consistent with a macrocytic type of anemia. Differential diagnosis is based primarily upon the history of previous surgery.

Sprue, nontropical sprue, and celiac disease all are characterized by steatorrhea. Sprue and nontropical sprue are found in adults while celiac disease is found in children. Classically, the stools are large, voluminous, frequent, greasy, and frothy. However, these findings are not consistent,[15] and as a result, very often the diagnosis is overlooked. The patient with these disease entities complains of fatigue, weight loss, weakness, abdominal distention, and cramps, diarrhea or constipation, and glossitis. There may be a history of tetanic contractions and symptoms of vitamin A, D, and B deficiencies. Anemia is usually present and may be either the macrocytic or microcytic type. Stool examination reveals large amounts of fatty acids and soaps. Serum calcium content is low and the oral glucose tolerance test curve is flattened. Diagnosis is made upon the basis of stool examination, serum calcium, and oral glucose tolerance curve.

Chronic pancreatitis may result in macrocytic anemia and may be confused with idiopathic steatorrhea. Differential diagnosis is based upon laboratory studies of stools, glucose tolerance curve, and pancreatic secretions. The stools contain a large percentage of free fats. The glucose tolerance test curve is usually high in chronic pancreatitis, and there is a significant decrease in the output of the pancreatic secretions.[16]

A diagnosis of regional ileitis is suggested by complaints of chronic colicky pain in the lower abdomen, fever, diarrhea, and emaciation. A mass may be palpated in the lower right quadrant. The diagnosis is usually made following x-ray examination with the appearance of a "string" sign.[17,18]

Chronic diarrhea may be an etiologic factor in the production of macrocytic anemia. Some of the disease entities producing chronic diarrhea have already been discussed, but there are many more. The differential diagnosis of chronic diarrhea necessitates a thorough and complete case evaluation.[19]

The anemia produced by the fish tapeworm, Diphyllobothrium latum, is identical to pernicious anemia.[20] This disease is found primarly in foreign countries, but has occurred around the lake region of the north central states. The symptoms are those of any tapeworm: vague abdominal pressure, colicky pains in abdomen, and irregular appetite. There may be nausea and vomiting with alternate periods of constipation and diarrhea. The icterus index of the blood is increased. Diagnosis is made upon finding segments of worms in the stool.[21]

Chronic liver disease must always be kept in mind when investigating macrocytic anemias because of faulty storage of erythrocyte maturation factor. The anemia is usually not severe and there is no poikilocytosis. The mean corpuscular hemoglobin concentration is normal and the bone marrow may or may not be megaloblastic.[22] Space does not permit lengthy discussion of differential diagnosis of liver disease. When it is suspected, various liver function tests should be made to help determine the presence and type of liver disease.

The achrestic type of macrocytic anemia produces only symptoms referable to the anemia itself.[4] There are no glossitis or central nervous system symptoms and the icterus index is not increased. Bone marrow and peripheral smears are of a megaloblastic and macrocytic character. Free hydrochloric acid is present in the gastric content and the chief point of differentiation apparently is the poor response to liver therapy and usually to folic acid. The clinical course of this type of anemia is chronic, progressive, and frequently fatal. Its pathogenesis is unknown but may be attributable to lack of the Will's factor or faulty utilization of the antianemic principle.

Macrocytic anemia may be found in hypothyroidism.[23] The systemic symptoms of pernicious anemia may be present as well as an achlorhydria, but the anemia usually is not as severe as pernicious anemia. There is usually little anisocytosis and no poikilocytosis, serum bilirubin is normal, and blood cholesterol is elevated. The metabolic rate is reduced and usually is the final determining factor in the diagnosis.[1]

In summary it must be emphasized that if a macrocytic type of anemia is found during examination of blood smears, bone marrow examination is an absolute necessity. Once megaloblastic anemia has been demonstrated numerous clinical entities must be differentiated as possible etiologic factors. Thorough clinical analysis is necessary to arrive at any definite differential diagnosis. However, it must be remembered that in some cases a therapeutic trial is necessary for final confirmation of diagnosis.

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PEDIATRICS—THEN AND NOW

In the present-day practice of medicine among children . . . it is estimated that the pediatrician spends over 50 per cent of his working time in various forms of preventive medicine, public health procedures, and education. The general practitioner is gradually becoming aware of his opportunities in these fields, and it is to be hoped that his interest will continue to increase, as it has been shown that at least 60 per cent of the children in this country receive all their medical care from the general practitioner. Although one third of his practice is devoted to the care of children, only 30 per cent of this service is devoted to the field of health supervision and preventive medicine.

As doctors increase their proficiency and knowledge along these lines, they will find a very satisfactory replacement for the many sick calls that their predecessors had to make in the earlier part of the century. They will also experience a deep sense of satisfaction as they recognize their ability to mold a child's physical and emotional progress so that it tends to become a healthier individual in succeeding generations. By their knowledge of the proper use of the new wonder drugs, doctors shorten the length of most illnesses, while they are taking an active part in the production of the ever-decreasing mortality figures. It is to be hoped that as their knowledge grows, some of the more unfortunate of the conditions mentioned above will gradually be conquered, or at least markedly decreased, by the physicians' expanding knowledge.—Editorial, *New York State Journal of Medicine*, January 1, 1953.

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ASPECTS OF GENERAL PRACTICE

The osteopathic profession has come to a point where its highest obligation is to understand the health needs of the nation and to fit itself to serve them. It is for these reasons that the successful launching of a College of General Practitioners within osteopathic medicine was hailed last March as a significant event in osteopathic history.[1] It is for these reasons that a review of the various aspects and responsibilities of general practice has been given detailed consideration in these columns.[1-3]

Last month THE JOURNAL presented a concise and explicit definition of a general practitioner, who would be capable of assuming diagnostic and therapeutic responsibilities (after hospital training) within certain limitations.[3]

This definition of a general physician is entirely applicable to the osteopathic general physician, providing such a practitioner holds osteopathy to be the practice of medicine and surgery within the guidance of the osteopathic concept. These broad and general principles which characterize the osteopathic approach constitute a guide for thought and action in all questions that relate to health and disease. Unless a physician applies to a thorough knowledge of medicine and surgery a critical and analytical evaluation based upon the osteopathic concept, he is not an osteopathic physician regardless of his degree.

The diagnostic responsibilities of the general practitioner find their basis in internal medicine, of which surgical diagnosis is an integral part. In this broad field almost half of the problems faced by the physician are generally classified as "functional" in nature. Doctors of medicine may dismiss such cases lightly, employ the placebo in some of its more modern forms, or place the condition in the newest diagnostic catch-all, the psychosomatic. Old-school medicine demands that a disease be named, refusing to recognize the disease process except it be demonstrable as an end-result. Doctors of osteopathy consider many conditions popularly classified as "functional" as possible manifestations of the disease process, not sufficiently advanced to be termed a definite disease, and even capable of reversibility if the proper therapy is employed.

Great credit, however, should be given old-school medicine for the emphasis it places upon thorough diagnostic routines. In the study of general practice in Volume 2 of the Report of the President's Commission[4] as well as in the report of Collings and Clark,[5] these routines are broken down into a primary and a secondary screening of patients.

The primary screening takes place in the doctor's office and consists of an initial history, a physical examination (to include blood pressure, rectal and/or pelvic examination), and laboratory findings (limited to a urinalysis, white blood count, and hematocrit). The osteopathic physician will ascertain the more obvious structural maladjustments as they relate to the patient's complaints.

The secondary screening can be continued in the office, but its completion may well necessitate hospitalization in the local institution. This procedure would consist of a more detailed history, physical examination, and laboratory studies. In more developed circumstances, the studies made may necessarily involve more difficult and less commonly used technics, in which the radiologist and pathologist will play an almost indispensable role. Consultation with specialists, especially in the fields of gynecology, dermatology, otorhinolaryngology, neurology, urology, orthopedics, and psychiatry, becomes an occasional necessity. The general practitioner bears a high level of diagnostic responsibility from which he cannot escape.

The experienced general practitioner will be able to solve the majority of his diagnostic problems by means of a carefully conducted primary screening. When the young physician arrives at a point where "he thinks like a doctor," he will acquire a nicety of judgment which will determine for him almost unconsciously to what degree of detail he must press his diagnostic routines. It can be confidently said that 80 to 90 per cent of the serious emergencies and potentially serious diseases seen in general practice can be diagnosed with reasonable accuracy by the qualified general practitioner.

Students of the role of general practitioner agree that he needs help much more from specialists in therapeutics than in diagnosis. They suggest, however, that the well-trained and experienced general

1. Editorial: College of general practitioners. J. Am. Osteop. A. 52:379-380, March 1953.
2. Editorial: Aspects of general practice. J. Am. Osteop. A. 52:436-437, April 1953.
3. Editorial: Aspects of general practice. J. Am. Osteop. A. 52:481-482, May 1953.
4. Building America's health: America's health status, needs and resources. Vol. 2. A report to the President by the President's Commission on the Health Needs of the Nation. U. S. Government Printing Office, Washington, D. C., 1952.
5. Collings, J. S., and Clark, D. M.: General practice, today and tomorrow. New England J. Med. 248:141, Jan. 22; 183, Jan. 29, 1953,

physician should be able to assume responsibility for the treatment of the great majority of conditions he diagnoses.

In the report[4] previously referred to, all responsibility for major surgery has been removed from the qualified general practitioner's scope of work, because there is general agreement that the demands of general practice prevent such a doctor from giving the time and attention necessary to a continuing mastery of major surgery and its advances. Conversely, a satisfactory general practice cannot be conducted if it is encroached upon by the demands of surgery. The general physician is limited, therefore, to minor surgery, which is defined "as nothing more than such procedures as the opening of superficial abscesses, the closing of minor wounds with simple sutures, and the biopsy of cervix and skin; all other procedures are regarded as major."[4]

This strict demarcation between minor and major surgery and the consequent limitation of the general practioner apparently do not take into account dire emergencies which demand immediate surgery regardless of the lack of a desired level of training of the physician. To meet this need, rural and semirural areas require general practitioner-surgeons capable of handling those catastrophic conditions that require immediate interference without sterile instruments if such are not obtainable, or that are emergencies but of such nature as to allow time for sterile instruments. It has been estimated that 40 to 70 per cent of all the surgery in the country is in the hands of the general practitioner-surgeon.

Pediatrics and obstetrics are two other specialties which bear a particular relationship to general practice. In modern times, the general practitioner has only begun to sense his newest responsibility in the care of children, the handling of behavior problems. In less populated areas, he must be prepared to assume every phase of pediatric care.

The general practitioner cares for the majority of the obstetric cases in the nation. He must familiarize himself with the unforeseeable emergency which lurks in almost every delivery and be ready to meet it. The general practitioner who assumes the responsibility of obstetric work should be able to meet such minimal requirements as the control of third-stage bleeding (short of hysterectomy), transfusion of exsanguinated mothers, making simple incisions, and applying low forceps. He should have available for consultation a qualified obstetrician, if only by telephone.

General practice as outlined by these four editorials is a challenge sufficient to command the lifetime effort of any young osteopathic physician. General practice is a challenge to our educational program, since so many osteopathic graduates enter general practice upon completion of their internships. It is a special challenge to fit the osteopathic approach into the broad program envisaged by this oldest field of medicine. The re-emergence of the general physician in the field of medicine is an indication of the validness of the family doctor whose supplantation by the specialist has been brief. This is not to decry the specialist in any field, but to assign him to his true status, which is above that of the fractionation of medicine.

In the past much criticism has been leveled at the general practitioner with some justification. The criticisms are well known, and they include that of attempting to care for too many patients to handle them well; that they do not "keep up"; and that they will try anything. Another common complaint is that the general practitioner does not refer cases "soon enough." All of these criticisms are not without merit, but all are answerable. It should be said that the potentialities of general practice have never been so great. It is not without a basis in fact that the Report of the President's Commission terms the general physician as the key man in a health service program adequate to meet a nation's needs.

Preparation of the general practitioner to fill his new role in medicine is a continuing education that starts in the undergraduate period and continues throughout professional life. The current educational technics of undergraduate medical education are ill adapted to the production of a fully qualified general practitioner, be they doctors of osteopathy or doctors of medicine. The casual postgraduate training of general practitioners is totally inadequate to keep them prepared to meet the demands made upon them.

Much can be done organizationally to raise the standards of general practice and to afford the general practitioner an opportunity for the continuous education so necessary for him if he is to measure up to his new opportunity and assume his broad responsibilities.

Herein is justification for the establishment of the American College of General Practitioners in Osteopathic Medicine and Surgery. At present it is our best means of providing academic and educational leadership. It can assist our colleges in training undergraduates for general practice in osteopathic medicine. It can take the lead in providing postgraduate training for general practitioners within the tradition and ideals of the "family doctor."

Complete justification for the College will be found to the degree that it successfully integrates the osteopathic concept into this newest responsibility of our profession—the development of the general practitioner within osteopathic medicine.

JOURNAL CHANGES

The "Classified Ads" which have hitherto been included in THE JOURNAL and in THE FORUM will, from this issue on, appear only in THE FORUM. As a service of the Association to osteopathic physicians, an attempt will be made to make the section more appealing and easily readable. The rates charged cover only actual printing costs.

The material formerly published in THE JOURNAL under the heading, "Official and Affiliated Organizations" will henceforth be carried in THE FORUM under the heading, "District and Local Groups." News

of national specialty groups will be reported in THE FORUM under the proper departmentalized heading.

Announcements of coming conventions and meetings and dates for state and national board examinations and reregistration of osteopathic licenses will continue to be carried only in THE JOURNAL. This material is not essentially news, but has important official and archival significance, hence the necessity of JOURNAL publication.

RECONSIDER

Osteopathic physicians who are planning to forego the Chicago convention in 1953 should reconsider while there is yet time. This Fifty-Seventh Annual Convention occurs at a high-water mark of osteopathic history—the recognition accorded this profession is wider than at any time in its history. The appearance on the program of the National Commander of the American Legion is valid evidence that the osteopathic profession plays an important role in the life of this nation. Such acceptance has ramifications that extend to the least known practitioner, and emphasizes the fact that every doctor of osteopathy, at least occasionally, should see his profession in action along its organizational and scientific front. And he should gain for himself that sense of belonging which comes as a direct result of meeting socially with his colleagues from every section of America.

There is no city in the United States more readily accessible than Chicago. If an after-convention vacation is possible, Chicago is the gateway to the great North or the far West. Summer days in Chicago beside Lake Michigan, for the Conrad Hilton overlooks the Lake, are delightful days. The Hotel itself is one of the world's great institutions and embodies every comfort for its guests, including air-conditioning in those rooms set aside for meetings and social events.

There may not be another meeting place so convenient for several years. And it is a place to bring the family for there are no members of any family for which this great city does not hold attractions. Ordinary "reasons" are not sufficient for not attending the Convention in 1953. Time, July 13 to 17; place, Chicago!

Notes and Comments

The Technical Bulletins published by the Veterans Administration have been recognized for some time as constituting a series of medical contributions of high order, characterized by practical brevity and high usability. They are distributed to all members of the American Medical Library Association and various groups of medical consultants in this country and abroad. Many of them could be obtained by osteopathic hospital libraries and should be made available to staff and student doctors in our hospitals approved for intern and residency training. . . . Among seven Technical Bulletins recently issued the following are especially significant: TB 10-76, Acute and Chronic Barbiturate Intoxication; TB 10-77, The Management of Peptic Ulcer; TB 10-81, The Management of Gastro-Intestinal Hemorrhage; and TB 10-82, Malignancy of the Colon and Rectum.

In TB 10-82 the reader's attention is called to the aversion of many doctors to making a digital examination of the rectum. Yet it is estimated that over 50 per cent of all cancers of the large bowel are within reach of the examining finger. This aversion is said to be so widespread as to render performance of such an examination the surgical consultant's chief function. A case at point was the patient recently seen at a large eastern hospital who had consulted four physicians regarding rectal symptoms, and was unable to get a digital examination. Finally, the patient made the digital examination and found a hard tumor mass located just inside the anus. Histologic examination following its removal proved the mass to be a carcinoma. . . . Carcinomas of the colon and rectum respond better to surgical therapy than malignancies located elsewhere in the gastrointestinal tract. Five-year survival rates run from 40 to 50 per cent in well-equipped clinics and the operative mortality is as low as from 1 to 3 per cent. This particular bulletin consists of 13 pages, covering such important topics as symptomatology and diagnosis, preoperative therapy, choice of an anesthesia, choice of operation, postoperative care, and the management of cases requiring colostomy, of precancerous lesions, and of patients with advanced malignant disease of the large bowel.

★ ★ ★

The principles of psychiatry should be applied by physicians as widely as those of chemistry and physiology—this in essence was the advice given by William C. Menninger, M.D., director of the Menninger Foundation, when he recently received an award for "an outstanding contribution to medicine." He cited the family doctor who once took time to listen to "the patient's story," in contrast to his successor who sees his patients in "highly professionalized offices" or "the impersonal wards of hospitals." Dr. Menninger termed it as fallacy to believe that a physician can prescribe "the total picture of incapacity of a social-psychological-biological unit strictly in terms of cellular changes. He proposed this question: "What shall we call the 'disease' represented by a man who has always been frail but has worked very hard to support his widowed mother, did not feel he could afford to get married,

buries himself in the details of a complicated job, develops paralyzing headaches, loses time at the office for which pay is deducted from his wages, worries about this so much that he loses sleep and begins vomiting after each meal? Just to make it complicated, he has a leukocytosis and an enlarged spleen." Dr. Menninger pointed out that psychiatric practice necessitates three diagnoses—"physical, psychological, and social." The modern physician, he contended, will recapture the effective assets of the old-time family physician by utilizing in daily practice the principles which govern psychiatric care.

★ ★ ★

When Hawaii comes into statehood it will be able to boast of a fine health record. Hansen's disease (leprosy, a term whose use is forbidden on the Islands) is no longer a problem. The famous Kalaupapa Colony of Molokai Island now has 234 patients in contrast to the 1,200 in 1910. Social stigma once attached to the disease is largely gone and patients are being returned to active life in the community. There have been no reported deaths from locally contracted smallpox since 1913, from diphtheria since 1946, or from typhoid since 1947. No typhoid has been reported in the last three years. Tuberculosis is now being brought under control. Chronic disease incidence is lower than on the mainland because of the lower average age of the population. Cooperation in a broad health program by public and voluntary health agencies and organizations has made Hawaii one of the healthiest places in the world.

★ ★ ★

Radioisotopes are accumulating in vast stores as waste products from atomic piles. The civilian employment of radioisotopes includes their usage as a drug, classifying as a "new drug" under the law. As such, they are subjected to carefully controlled tests under the supervision of experts of the Food and Drug Administration of the U. S. Department of Health, Education, and Welfare. Only after the safety of any new drug is determined, can it be distributed in interstate commerce for general therapeutic use. One firm is now producing and distributing iodine[131] to be used in the study and treatment of certain thyroid disorders. The Administration has control of drugs only in interstate traffic, but fortunately many states have comparable food and drug laws which apply to intrastate traffic as the Federal Act applies to interstate traffic. The value of the Federal Food, Drug, and Cosmetic Act to the health and welfare of the nation is again illustrated by the effective way in which it is being enforced by the Food and Drug Administration. It protects not only citizens, but safeguards as well the work of the doctor.

Proposed Amendments to the Constitution and Bylaws of the American Osteopathic Association

R. C. McCAUGHAN, D.O.
Executive Secretary

CONSTITUTION

(The following proposed amendment is published at the request of the Oklahoma Osteopathic Association and would make the Second Vice President and the Third Vice President members of the Board of Trustees with vote. The amendment was read in the House of Delegates in 1952 and can be acted upon by the House in 1953.)

Article VII—Board of Trustees and Executive Committee

Amend by inserting in the first sentence of the article, after the words "First Vice President," the words "Second Vice President, Third Vice President."

The Article would then read:

"The Board of Trustees of this Association shall consist of the President, President-Elect, the Past Presidents for the preceding two years, First Vice President, Second Vice President, Third Vice President, and of fifteen other members, five of whom shall be elected annually by the House of Delegates to serve for three years. The Board shall be the administrative and executive body of the Association and perform such other duties as are provided by the Bylaws.

"The Executive Committee of this Association shall consist of the President, Immediate Past President, First Vice President, President-Elect, the chairman of the Department of Professional Affairs and the chairman of the Department of Public Affairs."

BYLAWS

(The following proposed amendments are published at the request of Henry Turner, D.O., of New Zealand.)

Article II—Membership

Amend Section 6 by deleting in the second sentence the words "30 days" and substituting therefor the words "120 days." The sentence in question would then read: "After 120 days any objection to membership shall be certified to the Board."

Article II—Membership

Amend Section 6 by deleting in the third sentence the words "special and individual action may enroll the applicant," and substitute therefor the words "a three-fourth's majority may direct that the applicant be enrolled." That portion of the sentence would then read: "The Board of Trustees by a three-fourth's majority may direct that the applicant be enrolled as a regular member of the American Osteopathic Association . . . ."

Article II—Membership

Amend Section 6 by inserting in fourth line after the words "name of the applicant" the words "and the name of the unrecognized college of osteopathy from which he graduated." The clause in question would then read "thereafter, the name of the applicant and the name of the unrecognized college of osteopathy from which he graduated shall be published in the JOURNAL of the Association."

(The following proposed amendments are published to implement a recommendation of the Committee on Membership Approval presented to and approved by the A.O.A. Board of Trustees in July, 1952. These amendments would raise the dues of regular members during their second and third years after graduation, and during residency training.)

Article III—Fees and Dues

Amend the second paragraph of Section 1, by deleting the last sentence thereof, and by substituting therefor the following sentence: "Dues for regular members during the first, second and third years, immediately after graduation, shall be five dollars ($5.00) for the first year, twenty dollars

($20.00) for the second year, and thirty-seven dollars and fifty cents ($37.50) for the third year; i.e., all graduates from an A.O.A. approved college shall be entitled to only one first, second, and third year practice rate of five dollars ($5.00), twenty dollars ($20.00), and thirty-seven dollars and fifty cents ($37.50)."

Amend the third paragraph of Section 1, by deleting the paragraph and by substituting therefor the following paragraph:

"Dues for regular members serving an internship or residency in a hospital approved for intern or residency training by the American Osteopathic Association, shall be five dollars ($5.00) per year during internship and twenty dollars ($20.00) per year during residency training, regardless of when served."

(The following proposed amendment is published at the direction of the Board of Trustees of the Association to carry out a recommendation of the Committee on Membership Approval presented to and approved by the Board of Trustees in July, 1952. If the immediately preceding amendment to Section 1 of Article III—Fees and Dues, shall have been passed, the following proposed amendment will be unnecessary inasmuch as its content is included in the immediately preceding proposed amendment.)

Article III—Fees and Dues

Amend Section 1, in the third paragraph, by deletion of the phraseology, "i.e., all graduates from an A.O.A. approved college shall be entitled to only one first, second, and third year practice rate of five dollars ($5.00), twelve dollars and fifty cents ($12.50) and twenty-five dollars ($25.00)," and insertion of those words as a final sentence to paragraph two of Section 1 of Article III of the Bylaws.

(The following proposed amendment would leave to the direction of the Board of Trustees, by resolution, the details of the disbursements from the various funds of the Association.)

Article VII—Duties of Officers

Amend Section 4, paragraph (a) by striking out the words "upon the order of the Board of Trustees, signed by the President and Executive Secretary." and substitute therefor the words "under the direction of and in the manner prescribed by the Board of Trustees."

Department of Public Affairs

DONALD M. DONISTHORPE, D.O.
Chairman
Los Angeles

BUREAU OF PUBLIC EDUCATION ON HEALTH

HOBERT C. MOORE, D.O.
Chairman
Bay City, Mich.

JOINT COMMISSION ON ACCREDITATION OF HOSPITALS

In December, 1952, the Joint Commission on Accreditation of Hospitals assumed the responsibility for the hospital standardization program which had been conducted for 35 years exclusively by the American College of Surgeons. The creation of the Joint Commission on Accreditation of Hospitals followed a series of meetings between representatives of the American Medical Association, American Hospital Association, American College of Surgeons, and American College of Physicians. Its membership is composed of twenty members; seven are appointed by the American Hospital Association (one member representing the Canadian hospitals), six are appointed by the American Medical Association, three each are appointed by the American College of Physicians and the American College of Surgeons, and one is appointed by the Canadian Medical Association. The Joint Commission is the final authority for the accreditation of hospitals, carrying out the standardization program previously sponsored by the American College of Surgeons. *The Journal of the American Medical Association*, November 24, 1951, p. 1243, describes the Joint Commission as follows:

> The functions of the Commission will be: (1) to formulate standards relating to hospital accreditation; (2) to establish the type and scope of inspections to be made under this program; (3) to assign responsibility for hospital inspections to the several participating organizations; and (4) to award certificates of accreditation to qualifying hospitals. Inspections for purposes of hospital accreditation will be made and reported in a uniform manner by staff representatives of the participating organizations with the reports to be evaluated and acted on by the Commission itself. Inspections will be related primarily to the professional and administrative aspects of the hospital. They will not be concerned directly with the hospital's teaching program. Hospital surveys relating to internship and residency training will continue to be carried out by the organizations which have conducted these programs in the past. The operating expenses of the Commission will be paid by the constituent organizations on a basis proportionate to their representation on the Commission.

The first chairman of the Joint Commission on Accreditation of Hospitals is Dr. Gunnar Gunderson, M.D., chairman of the Board of Trustees of the American Medical Association. The Executive Director of the Commission is Dr. Edwin L. Crosby, M.D. Dr. Crosby graduated from the Albany Medical College in 1933 and prior to his appointment as executive director of the commission was the director of the Johns Hopkins Hospital at Baltimore. Dr. Crosby is the current president of the American Hospital Association.

In the first bulletin released by the Joint Commission to hospitals, it was explained that hospitals approved by the American College of Surgeons at the time of the transfer of the standardization program to the Joint Commission would remain on the Joint Commission's accredited list for the year 1953. It was stated that accreditation would be continued until an inspection of each hospital under the new Joint Commission program has been made. For the present, it was indicated the standards and point rating system developed by the American College of Surgeons would be used as the basis for accreditation by the Commission. Clause II of the Minimum Standards for Hospitals of the American College of Surgeons thus adopted requires in part, as one of the qualifications for membership upon a medical staff, that the physicians and surgeons be "graduates of medicine of approved medical schools, with the degree of Doctor of Medicine." The standards of the Joint Commission thus will prevent physicians and surgeons other than those possessing the degree Doctor of Medicine from serving on the staff of accredited hospitals.

In the year 1951, 2,740 hospitals were fully accredited under the general standardization program of the American College of Surgeons. The osteopathic profession has long protested the exclusion of its members from the staffs of some public hospitals solely because of the arbitrary standard of the American College of Surgeons. The first bulletin of the Joint Commission indicates that it will continue to enforce this requirement which has in the past prevented qualified physicians and surgeons not holding the degree Doctor of Medicine from serving on the staffs of hospitals. Some laws pertaining to hospitals or hospital services now refer to the hospital standards approved by the American College of Surgeons. In the future, it seems probable that some proposed laws or revisions pertaining to hospitals or hospital services may suggest that such hospitals conform to the minimum standards promulgated by the Joint Commission on Accreditation of Hospitals. Such proposals will probably have the support of all the membership organizations represented on the Joint Commission. Public health laws, however, should contain provisions which permit all qualified physicians and surgeons to contribute to the betterment of the public health. Standards of an organization such as the Joint Commission which are in contravention of this public purpose are not, therefore, in the public interest.

Department of Public Relations

CHESTER D. SWOPE, D.O.
Chairman
Washington, D. C.

BILLS IN CONGRESS

H.R. 1026—To amend the Public Health Service Act to provide medical, surgical, and dental treatment and hospitalization for certain officers and employees of the former Lighthouse Service. Favorably reported to House, House Report No. 319.

H.R. 2332—Requires annual review of military personnel requirements. Approved March 14, 1953, Public Law No. 7.

H.R. 3307—To provide for the treatment of users of narcotics in the District of Columbia. Passed House March 23, 1953.

H.R. 3375—Mr. Matthews of Florida. Amends internal revenue code to remove 5 per cent limitation regarding deduction for medical expenses.

H.R. 3434—Mr. Young of Nevada. Same as 3375.

H.R. 3469—Mr. Carnahan of Missouri. Amends internal revenue code to provide a deduction for certain expenses paid or incurred by the tax payer for his own education or for the education of any other person.

H.R. 3473—Mr. Doyle of California. To establish the Federal Agency for Handicapped.

H.R. 3487—Mr. Lane of Massachusetts. Removes self-employed exemptions from coverage of Federal old-age and survivors insurance.

H.R. 3488—Mr. Lane of Massachusetts. Permits voluntary coverage of self-employed lawyers under Federal old-age and survivors insurance.

H.R. 3554—Mr. Zablocki of Wisconsin. To amend the Social Security Act to provide disability insurance benefits for totally disabled individuals.

H.R. 3582—Mr. Hale of Maine. The National Health Act, 1953.

H.R. 3583—Mr. Hale of Maine. Provides for deduction of subscription charges to certain prepayment health service plans for the purposes of the federal income tax.

H.R. 3585—Mr. Javits of New York. Same as H.R. 3583.

H.R. 3586—Mr. Javits of New York. Same as H.R. 3582.

H.R. 3608—Mr. Kean of New Jersey. Extends coverage of Federal Old-Age and survivors insurance program, and increases the amount of earnings permitted thereunder without loss of benefits.

H.R. 3777—Mr. Perkins of Kentucky. Amends Social Security Act to provide disability insurance benefits for totally disabled persons.

H.R. 3779—Mr. Selden of Alabama. Amends internal revenue code to remove 5 per cent limitation against deduction for medical care.

H.R. 3850—Mrs. Bolton of Ohio. Nursing Education Act of 1953.

H.R. 3893—Mr. Johnson of California. Creating a National Manpower Board, for the most effective use of manpower resources of the United States.

H.R. 3911—Mr. Bolton of Ohio. Amends internal revenue code to liberalize deduction for medical care.

H.R. 3921—Mr. Miller of Nebraska. Regulates optometry in the District of Columbia.

H.R. 3930—Mr. Radwan of New York. Amends Longshoremen's and Harbor Workers' Compensation Act to permit injured employee to select his own physician.

H.R. 4128—Mr. Scott of Pennsylvania. National Health Act of 1953. Same as H.R. 3586, 3582, and S. 1153.

H.R. 4160—Mr. Kean of New Jersey. Amends Social Security Act to provide for a waiver of premiums to preserve insurance rights of permanently and totally disabled persons, and to provide rehabilitation of the disabled.

H.R. 4262—Mr. Donohue of Massachusetts. Creates Federal Agency for Handicapped.

H.R. 4393—Mr. Davis of Georgia. Amends internal revenue code to permit school teachers and other professional people to deduct, as business expenses, certain educational expenses.

H.R. 4417—Mr. Short of Missouri. Amends Army-Navy Nurses Act of 1947.

H.R. 4450—Mr. Edmondson of Oklahoma. Amends internal revenue code to reduce 5 per cent limitation to 2 per cent, against deduction for medical expenses.

H.R. 4456—Mr. Heller of New York. Authorizes Federal aid to cities for more efficient ambulance service.

H.R. 4495—Mr. Short of Missouri. Extends Doctor Draft Act until July 1, 1955. Passed House May 12, 1953.

H.R. 4593—Mr. Hagen of Minnesota. To assist voluntary nonprofit associations offering prepaid health service programs to secure necessary facilities and equipment through long-term, interest-bearing loans.

H.R. 4601—Mr. Teague of Texas. To clarify the law pertaining to hospital, domiciliary, and medical care of veterans.

H.R. 4642—Mr. Bosch of New York. Re-establishes emergency maternity and infant care program, for wives and infants of enlisted members of the armed forces.

H.R. 4676—Mr. Ford of Michigan. Amends Social Security Act to permit federal participation in public assistance for patients in private institutions for tuberculosis and for patients in private institutions for mental diseases.

H.R. 4689—Mr. Wolverton of New Jersey. Amends National Science Foundation Act of 1950 to remove limit on annual appropriations. Reported to House May 11, 1953. House Report 374.

H.R. 4785—Mr. Winstead of Mississippi. To authorize the interservice transfers of officers and enlisted persons of the Armed Forces.

H.R. 4981—Mr. Curtis of Missouri. Amends internal revenue code to remove $2500 and $5000 limitations applicable to deductions for medical expenses.

H.R. 5007—Mr. Teague of Texas. Relates to outpatient dental care for veterans.

H.R. 5016—Mrs. St. George of New York. Amends Food and Drug Act to identify aureomycin by its chemical name, chlortetracycline.

H.R. 5017—Mr. Short of Missouri. Introduced May 5, 1953. To amend the Army-Navy-Public Health Service Medical Officer Procurement Act of 1947, as amended, so as to provide for appointment of doctors of osteopathy in the Medical Corps of the Army and Navy.

Be it enacted by the Senate and House of Representatives of the United States of America in congress assembled, That section 201 of the Army-Navy-Public Health Service Medical Officer Procurement Act of 1947 (61 Stat. 777), as amended, is further amended by inserting immediately after the word "medicine" wherever used therein, the words "or osteopathy."

Sec. 2, Section 201 of the Army-Navy-Public Health Service Medical Officer Procurement Act of 1947 (61 Stat. 777), as amended, is further amended by adding the following at the end thereof:

"A doctor of osteopathy to be eligible for appointment in the Medical Corps of the Army and Navy must be a graduate of a college of osteopathy whose graduates are eligible for licensure to practice medicine or surgery in a majority of the States, and be licensed to practice medicine, surgery, or osteopathy in one of the States or Territories of the United States or in the District of Columbia."

S. 1068—Mr. Griswold of Nebraska (for himself and Mr. Ives of New York). To provide hospital care and treatment for certain disabled veterans.

S. 1153—Mr. Ives of New York (for himself and Mr. Flanders of Vermont). National Health Act of 1953.

S. 1154—Mr. Ives of New York (for himself and Mr. Flanders of Vermont). Provides for deduction of subscription

charges to certain prepayment health service plans for the purposes of the Federal income tax.

S. 1215—Mr. Hunt of Wyoming. To create Department of Health, Education, and Public Welfare.

S. 1251—Mr. Mundt of South Dakota. Amends Draft Act relating to veterans' exemptions.

S. 1356—Mr. Kefauver of Tennessee. Amends Social Security Act to provide for payment of child's insurance benefits under OASI to children 18 years of age incapable of self-support because of physical or mental disability.

S. 1436—Mr. Griswold of Nebraska. To establish a Federal Board of Hospitalization.

S. 1470—Mr. Kefauver of Tennessee. To provide for voluntary coverage under the Federal old-age and survivors insurance system for lawyers and doctors engaged in the practice of their professions.

S. 1495—Mr. Murray of Montana. Re-establishes emergency maternity and infant care program for wives and infants of enlisted members of the Armed Forces.

S. 1514—Mr. Taft of Ohio. To establish a Commission on Governmental Functions and Fiscal Resources. Commission will study and investigate and determine and report whether there is justification for Federal aid in the various fields in which Federal aid is extended, whether there are other fields in which Federal aid should be extended, whether Federal control with respect to these activities should be limited, whether Federal aid should be limited to cases of need, and other matters incident to Federal aid, including the ability of the Federal Government and the States to finance activities of such nature. Passed Senate May 6, 1953. Title changed to Commission on Intergovernmental Relations.

S. 1515—Mr. Hunt of Wyoming and others. Permits compact by certain Western States relating to higher education.

S. 1526—Mr. Saltonstall of Massachusetts. Provides for appointment of Chief of Medical Service Corps of the Navy.

S. 1530—Mr. Saltonstall of Massachusetts. Amends Army-Navy Nurses Act of 1947.

S. 1531—Mr. Saltonstall of Massachusetts (for himself and Mr. Hunt of Wyoming). Extends Doctor Draft Act until July 1, 1955. Same as H.R. 4495. Mr. Hunt also introduced amendment to S. 1531 to extend $100 per month extra pay provision of Career Compensation Act to volunteer medical and dental officers to July 1, 1955.

S. 1551—Mr. Flanders of Vermont. Establishes National Manpower Board.

S. 1584—Mr. Murray of Montana. National Leprosy Act

S. 1748—Mr. Taft of Ohio. To incorporate the National Fund for Medical Education.

S. 1789—Mr. Sparkman and Mr. Hill of Alabama. To provide for the disposal of certain private hospitals, clinics, and medical facilities acquired by the VA.

S. 1834—Mr. Young of North Dakota. Amends Selective Service Act to permit correction of dental defects before induction.

RENEW FEDERAL NARCOTIC AND MARIHUANA PERMITS

Osteopathic physicians and osteopathic colleges or research laboratories must renew their Federal narcotic permits every year before July 1. Failure to do so entails severe penalties.

On or before July 1 each osteopathic physician who wishes to dispense, prescribe, or administer marihuana in his practice must, under the provisions of the Federal Marihuana Tax Act, pay a $1.00 annual tax and register or reregister with the Director of Internal Revenue for each of the districts in which he practices.

Current Medical Literature

AN EVALUATION OF NEEDLE BIOPSY OF THE LIVER

In an article appearing in the *American Journal of Medicine*, December, 1952, Edward R. Christian, M.D., reviews the results of 100 needle biopsies of the liver performed on 89 patients who had evidence—historical, clinical, or laboratory—of liver disease.

Approximately 60 per cent of the biopsies were performed without preoperative sedation, and in the remainder the sedation was mild, usually consisting of small doses of Demerol.

The Vim-Silverman needle was used throughout the series. The obturator was not used and the sleeve needle and split cannula were inserted as a unit. The insertion site was the eighth or ninth intercostal space in the right midaxillary line. The speed of the operation is indicated by the fact that patients were not permitted to breathe during the procedure. Successful biopsies were obtained in 98 per cent of the cases.

The complications in the author's patients were few and minor. Only 25 required pain relief, 1 suffered from nausea and vomiting, and hemorrhage developed in an uncooperative alcoholic who disobeyed orders. It is possible to hospitalize the patient, perform the biopsy, and discharge the patient with safety within 24 hours.

The procedure was of greatest value in the problem of differential diagnosis and in making an accurate diagnosis which was not obtainable from clinical data.

The main objection to needle biopsy is its failure to show isolated or widely separated lesions of the liver. However, of 14 patients subjected to biopsy on whom autopsies were later performed, in only one instance did the two findings not agree. A study was performed to demonstrate the ability of needle biopsy to show the histology of the entire liver. In a series of 10 autopsy cases, submitted without identification to the pathologist, biopsy findings and findings from examination of the entire liver generally agreed.

The author disagrees with the objection that needle biopsy specimens are too small for histologic staining technics. He has found that one biopsy specimen can supply 45 to 60 sections for microscopy for as many as nine staining procedures and that these specimens withstand the technics without disintegration.

THE MANAGEMENT OF ANGINA PECTORIS

Angina pectoris is defined by Maurice M. Best, M.D., and J. Murray Kinsman, M.D., in the February, 1953, issue of the *Journal of the American Geriatrics Society* as a syndrome due to decreased caliber or partial closure of the coronary artery lumen brought about by arteriosclerotic or nonarteriosclerotic changes.

Before management can be considered, a correct diagnosis is necessary. History and an abnormal electrocardiographic reaction are the two important factors in diagnosis. However, an anginal patient at rest frequently presents a normal electrocardiogram. Test conditions have been devised in which electrocardiographic abnormalities appear. These results have proved useful in diagnosis and treatment.

The objectives in treating angina pectoris are twofold: (1) treatment and rapid termination of the pain attack and (2) prophylactic measures to reduce the number of attacks and their severeness.

The authors stress the importance of explaining the condition to the patient. The term "coronary insufficiency" is used in offering a sympathetic explanation to the patient. The aim is the prevention of a cardiac neurosis and unnecessary invalidism.

Any associated disease conditions should be corrected since correction may result in discontinuance or reduction of

the symptom complex. The importance of watching the diet and keeping the weight to a "low normal" level is emphasized to the patient. Mortality from angina pectoris is twice as high in overweight persons.

Chest discomfort is the criterion for permission to exercise. If no chest discomfort is present, walking on level ground is allowed, except immediately after eating a large meal or in extreme cold.

The patient is requested to keep a diary in which each attack, the number of attacks, and the conditions bringing on each attack are recorded. The physician uses this daily diary as a treatment guide.

The drug treatment of angina pectoris lists the vasodilators and other drugs used or in the experimental stage. Nitroglycerine is recommended as the most valuable drug for relief. It can also be used as a preventive before exposure to stressful situations. The drug is safe, and there are minimal side effects.

Khellin has been found to be quite helpful in a small percentage of patients but the side effects are frequent. However, investigation shows that combined intramuscular and oral administration or oral use with Resion do not appear to be followed by the same effects that follow oral administration alone. Pentaerythritol tetranitrate is not recommended for all types of angina pectoris, but the incidence of toxic side effects is lower than with khellin. Dioxylline phosphate is useful in some cases and has few side effects. Other drugs sometimes used are heparin, radioactive iodine, and phenobarbital, but the first two have not been fully evaluated as yet.

Surgery is used for patients who do not respond to conservative therapy. Pain-abolishing procedures are used. Posterior root ganglia from the first to sixth thoracic vertebrae are injected with 95 per cent alcohol, posterior root ganglia from the first to fifth thoracic are sectioned, or upper thoracic sympathetic ganglionectomy is performed.

CONGENITAL HEART DISEASE IN CYANOTIC CHILDREN

Because of increased publicity given to the subject of "blue babies" in the past few years, most mothers noticing any signs of cyanosis in their children immediately seek medical attention for them. This cyanosis frequently is the only outward sign of congenital heart disease. In an article appearing in the February, 1953, issue of *California Medicine*, Willis J. Potts, M.D., gives some of the more common cardiac abnormalities that can be diagnosed from cyanosis and other symptoms.

Squatting after unusual exertion should make the physician suspicious of tetralogy of Fallot. Great incapacitation and mild cyanosis are indicative of pure, or isolated, pulmonary stenosis. A systolic murmur which is brisk, short, and rasping usually may be heard in tetralogy of Fallot, pure pulmonary stenosis, and tricuspid atresia. The erythrocyte count ranges from 5 to 11 million per cubic millimeter in cyanotic children. The hemoglobin content of the blood is also high.

The heart in tetralogy of Fallot is, as a rule, boot-shaped, but it may be normally shaped and usually is of a normal size although it may occasionally be slightly larger than normal.

Pure pulmonary stenosis has symptoms which are like those of tetralogy of Fallot, but the pathologic features and therapy are not at all similar. X-rays and electrocardiograms may show diagnostic features.

Tricuspid atresia has symptoms which are similar to those of tetralogy of Fallot. Cyanosis, a systolic murmur, round heart, left deviation of the axis in electrocardiograms, and clear lung fields usually point to tricuspid atresia.

The question is raised as to when cardiac catheterization should be done and angiocardiograms made. The author does not think they should be used unless a definitive diagnosis cannot otherwise be made. The procedures are valuable but not necessarily conclusive, and angiocardiography can be dangerous.

For tetralogy of Fallot, the author prefers the aortic-pulmonary or subclavian-pulmonary anastomosis, depending on the curve of the aortic arch. If the condition of the child permits, operation should be postponed until the child is at least 3 years old, since the mortality rate for children under 3 years is 16 per cent and for those being operated on after the age of 3 the mortality is 2.5 per cent.

In pure pulmonary stenosis the pulmonary valve is opened with an expanding valvulotome after which it is dilated.

The treatment of choice for tricuspid atresia is a shunt operation, but the additional strain on the heart may later lead to cardiac failure.

Even though all patients do not do well after operation, Potts feels that the effort is worth while because so many of the children improve tremendously and are able to lead lives more nearly normal.

THE INFLUENCE OF MODERN ANTIBIOTICS ON ABDOMINAL SURGERY

Although the antibiotic era is less than 10 years old and a widening of the field of abdominal surgery has taken place in that time, the antibiotics cannot be considered to be the cause of any great change in this area. In an article appearing in the *New York State Journal of Medicine*, January 15, 1953, Roswell K. Brown, M.D., discusses how abdominal surgery has been little affected by the use of antibiotics.

Abdominal surgical diseases are for the most part not of a bacterial or infectious origin. Although appendicitis mortality rates have declined steadily in the past 2 decades, it is doubtful that much of the improvement can be accredited to the use of antibiotics.

The author believes that there is a tendency toward substitution of antibiotics for proper technics and that this is one of the greatest dangers of their use in abdominal surgery.

He classifies the uses of antibiotics into (1) systemic administration (of antibiotic to which invading organisms are sensitive) in treatment of established infection and (2) prevention of infection with antibacterial agents which are not absorbed into tissues. This classification is based upon a definition of infection as bacterial invasion of tissue and of contamination as the presence of bacteria in or on a wound.

If there is no accurate bacterial diagnosis which can be used as a therapeutic guide, there should be limited use of antibiotics. When pus is encountered during operation, an antibacterial should be used aseptically to prevent contamination.

There is no unanimity among surgeons as to indications or time for operation, even with the use of antibiotics. A differentiation between contraindications to surgery is necessary —they are based on the patient's condition, the possibility of infection complicating the operation, and the possibility of inflammation causing technical difficulties. It is the opinion of the author that operation should not be postponed because of acute inflammation. A case report is given in which an appendectomy was performed on a 6-year-old girl 17 days following drainage of an appendiceal abscess and treatment with penicillin and aureomycin. She recovered rapidly and left the hospital 7 days after the appendectomy. Since the current practice is postponement of appendectomy for 2 months following appendiceal abscess drainage, in view of this experience it is suggested that the problem be restudied for determination of the optimum time interval.

Systemic use of antibiotics routinely for prophylaxis in abdominal surgery appears undesirable; with such use there is always danger of toxic reactions to the antibiotics. In contamination, preventive topical use of antibiotics seems warranted.

In a discussion of the article, R. Sterling Mueller, M.D., disagrees with the statement that only a small part of the improvement of the mortality rates in acute appendicitis is due to the use of antibiotics. He points out that in New York City there were three times as many deaths from acute appendicitis in 1941 as there were in 1951. He also approves of the practice of using antibiotics prophylactically in clean or relatively clean operations, stating that the few reactions that occur are not usually serious.